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No. 1

HISTOPATHOLOGIC STUDY ON TWO CASES OF "CENTRAL NEURITIS"; DEMONSTRATION OF A NEW GRANULE ("NUCLEOPROTEID-LIKE GRANULE") IN THE NEUROGLIA CELLS *

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INTRODUCTION

Since Dr. Adolf Meyer¹ described, in 1901, the symptom-complex "central neuritis," which he introduced as a short designation of the "parenchymatous systemic degeneration mainly in the central nervous system," a number of cases have been added to the literature by different authors, and there is no doubt that this condition can be recognized from its clinical symptoms and established anatomically. According to the descriptions of authors, this symptom-complex is found to occur in depressive psychoses at the time of involution, in alcoholic senile states and in alcoholic phthisical-cachectic states, and the clinical condition is characterized by diarrhea, emaciation, twitchings and rigidity of the extremities, some fever and some changes in the reflexes, and the mental condition is an anxious, perplexed agitation, delirium or stupor. Anatomic investigation revealed "changes somewhat similar to those found in the motor cells of the cord and the medulla, from which the nerve process has been cut off at the periphery" in the Betz cells and other cells, and systemic degenerations of the central nerve fibers. Beside the accurate description of Dr. Meyer, there are a number of excellent histologic studies on changes of the nerve cells; however, we find relatively few investigations on alteration of the neurofibrils of nerve cell as well as the neuroglia tissue. The material of the Phipps Clinic recently offered the opportunity to study two cases of "central neuritis." The alteration of the intracellular neurofibrils of nerve cells as the effect of cutting off the nerve process has been a topic of several experimental studies. In view of the clear relationship of the changes of the nerve cells in

* From the Neurological Laboratory of the Henry Phipps Psychiatric Clinic, the Johns Hopkins Hospital, Baltimore.

1. Meyer, Adolf: On the Parenchymatous Systemic Degeneration Mainly in the Central Nervous System, Brain, Lond. 24: 1901.

"central neuritis" and the axonal alteration resulting from section of fiber, an investigation of the appearance of the neurofibrils may be of value, as "central neuritis" is, so to speak, an experiment of nature, although the nerve process is not really cut off. Thorough investigation of the changes of the neuroglia tissue led me to demonstration of a new granule in neuroglia cells, a finding which may throw a new light on the problems of the metabolism and catabolism process of the central nervous tissue.

Before taking up the description of my investigation, I beg to express my highest appreciation of the kindness of Dr. Adolf Meyer, who gave me the privilege of studying these cases and many other specimens of our laboratory, and also gave me many useful suggestions in this study.

ILLUSTRATIVE CASES

CASE 1.—Clinical History.—E. B., a housewife, aged 29, in January, 1918, presented the skin lesion of pellagra, constipation, epigastric distress, and restlessness. Admitted to the medical service May 25, she showed marked emaciation and peculiar very dark pigmentations over the entire body. She became delirious May 29, and was transferred to our clinic. She developed a stupor and a typical picture of "central neuritis," with tenseness, rigidity, tetanoid positions and jactitations, and a terminal temperature of 107 F.

Family History.—A sister has nervous headaches, the daughter has tantrums and night terrors; an aunt broke down after the death of her husband.

Personal History.—The patient had the childhood diseases, typhoid fever at 16 and rheumatic fever at 20. Her development was abnormal; she married at 19 against her parents' wishes. Her married life was uneventful except for a miscarriage at 20 and an abortion at 28. The patient's habits have been regular, but the diet consisted mostly of vegetables. She has two children.

Present Illness.—In November, 1916, she began to become irritable. A spontaneous abortion in March, 1917, with hemorrhage for three months increased this condition and weakened her. She recovered somewhat during the summer, but in September, 1917, restlessness, night sweats, and her old symptoms returned. By December, 1917, she was worse, easily upset and much worried when her younger child developed scarlet fever. In January, 1918, epigastric distress, giddiness, flashes of light, and roughening of the hands appeared, and grew gradually worse. She took to bed on March 13; two days later her temperature was 101 F. and she had difficulty in swallowing. The last two symptoms left her March 16, and on light diet and rest she gradually improved till May 8, being out of bed she showed thirst, tachycardia, sleeplessness and a return of the other symptoms. On May 25 she was admitted to the medical service of the Johns Hopkins Hospital, with her hands in the condition described. Her mouth and tongue were sore; she was emaciated, very weak, slightly anemic, and her skin was dry and darkly pigmented. She was mentally clear until May 29, when she had an episode of screaming and confusion. On May 30 she was at times disoriented, frightened and was incontinent. On May 31 she was completely disoriented, delirious and incontinent. Her temperature was 101.5 F., and the pulse 140.

Mental Status.—The patient lay in bed twisting her hands and face, picking at the bed clothes and moving her jaw frequently. She mutters unintelligibly. Her arms and legs are rigid in flexion. She is usually unresponsive. She is completely disoriented and in the medical service had imagined she was giving birth to a baby. Incontinence.

Physical Status.—The patient is extremely emaciated; her skin harsh and dry and diffusely pigmented. On the backs of her hands and front of wrists and on the knuckles there is a dry, cracked, raised, very dark thickening of the skin symmetrically distributed. She is pale, her tongue red with swollen papillae; pyorrhea and dental caries. Tenseness and rigidity of musculature, and limbs in flexion, very hyperactive deep reflexes.

Progress in Clinic.—After admission in our clinic the patient grew continually worse; she muttered to herself, became more rigid and more restless, slept none and appeared to have slight spasms with retraction of the head. Later she was unable to swallow. Her temperature rose to 107 F., on June 2. Her pulse rose to 180 and Biot's breathing appeared. She died June 3.

Necropsy.—No macroscopical lesion in the brain and in the spinal cord; the microscopic study revealed "central neuritis."

CASE 2.—Clinical History.—A. S., widow, aged 48. Dating from September, 1917, the patient was nervous and weak. Dilatation and curettement operation followed by delirious state. On admission (December, 1917) the patient showed amebic dysentery with suspicion, hallucinations of hearing, disorientation, delusions and fear. This mental state kept pace with her physical dilapidation, which rapidly led to exitus.

Family History.—Unimportant.

Personal History.—Negative. The patient felt "run down" and weak for the past year, without, however, any failure in business activities. In November, 1917, the patient appeared very weak; kept in bed for two weeks and on Nov. 17, 1917, a D. and C. operation was done with prolonged response to anesthetic. Following the operation the patient was agitated and suspicious of food. When she tried to jump out of the window she was transferred to a sanitarium. The patient developed diarrhea, six to seven stools a day, watery and bloody; she was very weak and during the last three days prior to admission she was tube-fed.

On admission the patient seemed confused and disoriented and profoundly exhausted. For a time she was restless, showing sudden paroxysms as though in pain. The knees were flexed and the abdominal muscles felt contracted. During the attacks she rolled from side to side in the bed; but between the attacks she seemed relaxed and quiet.

Physical Status.—The patient is a profoundly undernourished, emaciated anemic-looking woman. The neurologic examination showed definite exaggeration of deep reflexes. The abdomen is boat-shaped; the patient holds the muscles tense. Stool examination revealed the existence of *Ameba histolytica*. The patient began to run a temperature after three days in the hospital, ranging between 99 and 101 F.

Mental Status.—The patient thought her food was poisoned and she refused food, so that tube feeding was at times required. She also sometimes talked vaguely of being married to a Dr. Adams. She also had auditory hallucinations. The patient was usually oriented to time and place, but had difficulty in getting names straight. Retention and memory for remote past were good. She showed no definite affective disturbances.

Progress in the Clinic.—The diarrhea continued throughout the course in the hospital with from one to seventeen stools a day. The temperature ranged between 99 and 101 F. The above mentioned mental state continued till the end of January, 1918, interrupted now and then by remissions, in which she was fairly well oriented and showed no delusions. On January 31, marked twitchings of facial muscles and tremor of the lips were noticed.

From the beginning of February, 1918, the patient's condition appeared worse; the weakness increased and nausea and vomiting came on after meals. Defecation and micturition often occurred involuntarily. On February 13 the temperature rose from 101 to 104, and the pulse to 140. The patient became semicomatose and died.

Necropsy.—The brain showed superficially normal findings; microscopic study established typical "central neuritis."

MICROSCOPIC FINDINGS

(A) *Manifestations of the Nerve Cells.*—In both cases of "central neuritis" the paracentral lobule and the anterior central gyrus were subjected to a most extensive study. The spinal cord was examined only in the first case. To avoid repetition I shall describe below the findings of both cases together, and besides mention only points of difference between two cases.

We shall begin with the description of the findings in thionin preparations. So far as the cytoarchitecture of the cortex is concerned, both cases showed nothing abnormal. Almost all the Betz cells are in both cases in a more or less advanced stage of axonal reaction. The cell body is more or less swollen, and shows in a certain place an area of an almost homogeneous character due to a decay of the stainable substance into dust-like particles. In the extremely altered cell, the latter looks like a more or less deeply blue stained balloon. In the second case we find fairly well preserved stainable portions in the dendrites and also lumps of the Nissl bodies at the periphery of some cells, forming a rim around the margin. The "whirlpool" arrangement of the Nissl bodies, described by Dr. Meyer,^{1, 2} did not come to our observation in this case. In the first case the superimposed fever alteration complicated the characteristic features; even at the periphery of the cells and in the dendrites the Nissl bodies have disappeared and the protoplasm is stained diffusely blue. The nucleus is found more or less dislocated in every cell usually to the side or toward the neighborhood of apical dendrite, causing sometimes a projection of the cell surface. The nucleus itself is sometimes round, sometimes it looks elongated or distorted and flattened, as if pressed to the wall by the swollen portion of the cell. The nuclear membrane is usually sharply marked, the content is pale,

2. Meyer, Adolf: Demonstrations of Various Types of Changes in the Giant Cell of the Paracentral Lobule, *Am. J. Insan.* 54: 1897.

the nucleolus is round and generally deeply stained. Along the margin of the swollen cell or sometimes accumulated in one point of the cell there appears a heap of yellowish, greenish pigment. Figure 1 was taken from a Betz cell of the second case.

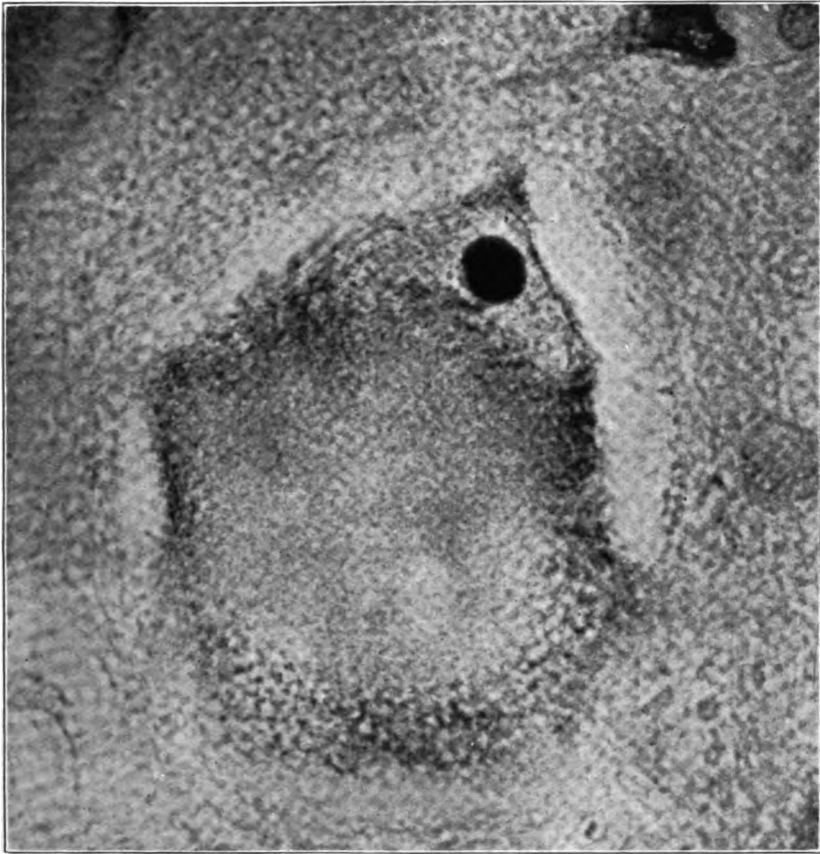


Fig. 1.—A Betz cell from the second case.*

In our first case some of the pyramidal cells of outer layers also show axonal reaction; however, most of them give an entirely different picture. They are mostly swollen; the dendrites are also enlarged. The Nissl bodies had largely disappeared from the cell body; no dislocation of the nucleus was noticed. The whole picture corresponds to that which Goldscheider and Flatau described as the effect of arti-

*Figures 1 to 6 are photomicrographs and Figures 7 to 9 were drawn with the use of the Abbe camera lucida with slight rotation of the micrometer screw.

Figures 1, 5, 6, 8 and 9 are thionin preparations.

ficial increase of body temperature. We also found some shrinkage cells (so designated by Hoch³) in the second and third layers.

In the second case the medium-sized and small pyramids also show a close resemblance to Hoch's shrinkage cells; the "alteration with rarefaction around the nucleus" of Hoch was also found in some of the larger pyramids. The thionin preparations of alcohol material show practically the same picture as the above described preparation of formalin material.

A portion of the motor cells of the spinal cord and some of the cells of the Clark column show in our first case typical axonal reaction, although in less advanced stages, and some of them show, moreover, the fever alteration. Some other cells show merely fever alteration.

The manifestations of the intracellular neurofibrils of the nerve cells were studied in Bielschowsky preparations. Cotton and Southard⁴ found in their Cajal and Bielschowsky preparations a definite change in the fibrils within the chromatolyzed area. Lambert⁵ observed a disappearance of the fibrils exactly corresponding to the area of swelling in the Nissl equivalent. Figure 2 shows the alteration of the fibrils and the nerve cell; with the lower magnification the Betz cells appear as swollen bodies with sharply marked dendrites, and thus reminds us of the bed-bugs. In higher magnification we can follow different stages of the neurofibril alteration in different cells. The fibrils are in more or less advanced stages of fragmentation, and in the extreme state they are dissolved to fine dust-like pieces. In the central part of the cell body, where thionin preparations show an homogeneous area, we find also in the Bielschowsky stain a glossy area; however, at the periphery of the cell body and also in the dendrites, in which thionin stain disclosed well marked Nissl bodies, we find fairly well preserved neurofibrils, sometimes running from one dendrite to another. In the apical dendrite of the Betz cells the neurofibrils are especially clearly demonstrated and run isolated or in bundles toward the cell body. It is a striking feature that these neurofibrils end sharply at the base of the dendrite. These findings are very interesting, as they tend to indicate that the alteration of the neurofibrils keep pace with the dissolution of the Nissl bodies. As a

3. Hoch, August: Nerve Cell Changes in Somatic Diseases, *Am. J. Insan.* **55**: 1889.

4. Cotton, H. A., and Southard, E. E.: A Case of Central Neuritis with Autopsy, *Am. J. Insan.* **65**: 1908.

5. Lambert, C. I.: Notes on Pathological Material, *N. Y. State Hosp. Bull.* **1**: No. 3, 1908.



Fig. 2.—Bielschowsky stain; a Betz cell from the second case. The nucleus is found on the left-hand side of the cell; the pigment is found on the right-hand margin of the cell.

further evidence of this phenomenon may serve the Bielschowsky preparation of our first case; here we do not find any neurofibrils in most of the Betz cells even at the periphery of the cell body and the dendrites and thionin stain disclosed, as already remarked, dissolution of the Nissl bodies in the same. This corresponds to the statement of Lambert,⁵ who observed more or less apparent fragmentation and granulation of the fibrils in the fever change. Marinesco⁶ stated that the changes in the neurofibrils and the chromatophil elements as a rule occur and progress together. Dr. Meyer⁷ and Lambert consider the changes in the fibrils almost invariably coincident with the changes of the local protoplasm. The Bielschowsky preparation of the spinal cord in our first case brought out samples of axonal reaction in the anterior horn and the Clark column; but owing to the superimposed fever alteration the neurofibrils are marked only in a few cells at the cell periphery and in the dendrites.

(B) *Manifestations of the Nerve Fibers and the Axis-Cylinders.*—The Marchi preparations showed definite myelin-sheath degeneration in the marrow of the paracentral lobules and the anterior central convolutions; however, the degenerated myelin-sheaths are so scanty that only a few appear in one Marchi section in both of our cases (Fig. 3). The Marchi cross-section of the spinal cord of our first case shows a number of degenerated fibers in the anterior and lateral pyramidal (Fig. 4) bundles as well as the cerebellar afferent bundles; but the degeneration in the pyramidal bundles is much slighter than the findings in the paracentral lobule and the anterior central convolution would make one expect; Dr. Meyer¹ and others also noticed this incongruity.

The Alzheimer-Mann preparations of the anterior central convolutions disclosed a very interesting finding in both of our cases (Fig. 7). In this preparation normal axis-cylinders are brought out in a deeply blue stain; but among those we find axis-cylinders with red-stained parts of different length in their course. Some fibers show colorless parts instead of the red-stained part. Between the red-stained part and blue-stained part of the fiber we find sometimes a very short colorless part, and sometimes we observe that the red-stained part is interrupted by colorless parts in many segments. Some axis-cylinders have in their course more than one red-stained or colorless part, so that they look like strings of changing color. It admits no doubt

6. Marinesco: Lesions of the Neurofibrils in Certain Pathological Conditions, *Rev. Neurol and Psychiat.*, 1905.

7. Meyer, Adolf: Discussion of the Data Concerning Neurofibrils, with Illustrations, *Proceedings New York Neurol Soc.*, Feb. 6, 1906. *J. Nerv. & Ment. Dis.* **33**: 1906.

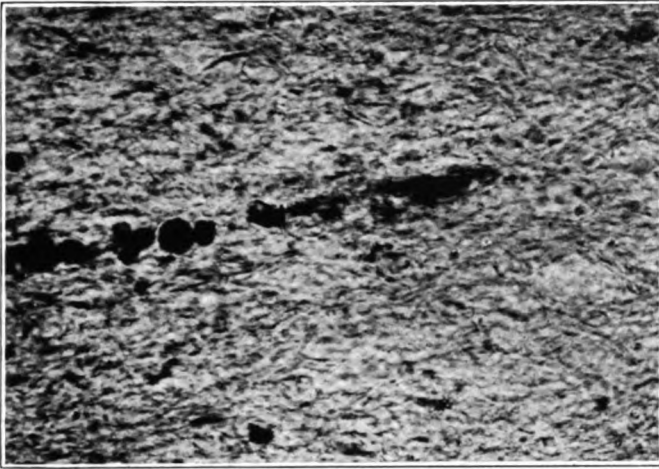


Fig. 3.—Marrow of the anterior central convolution (first case). Marchi's stain.

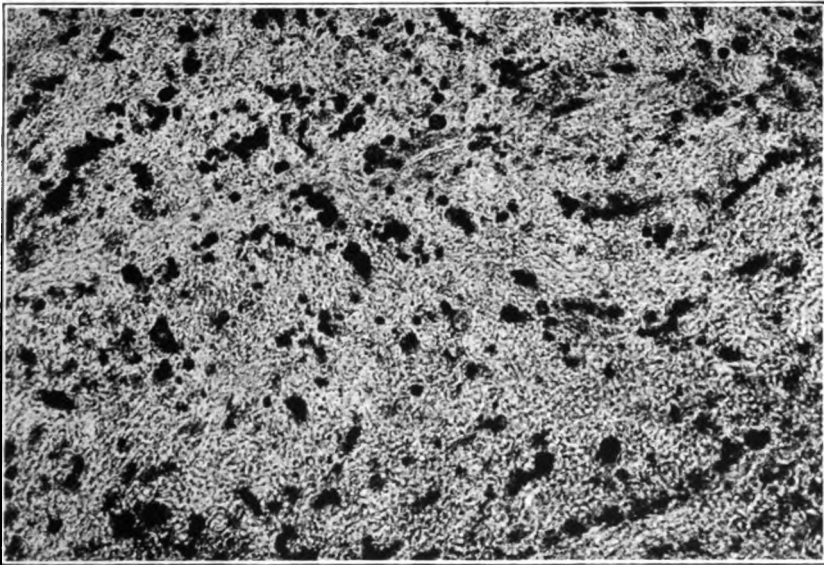


Fig. 4.—Cross-section of the lateral pyramidal bundle (first case). Marchi's stain.

that these red-stained and colorless segments of the fibers are degenerated, and that the red-stained parts are in a more advanced state of alteration than the colorless parts. Now it is worthy of note, that these degenerated axis-cylinders were found in the sections much more numerous than the degenerated myelin-sheaths demonstrated by the Marchi method. The Alzheimer-Mann stain of the longitudinal sections of the cord in our first case revealed many such degenerating axis-cylinders in the lateral pyramidal bundles and in the cerebellar afferent bundles.

On the basis of the fact, that in acute cases the cell change far outweighs the fiber degeneration, Cole⁸ claimed that the former are almost certainly primary, and the latter appear to be simply a secondary manifestation of the former. But considering the possibility that the degeneration of the myelin sheaths may be demonstrable by the Marchi method in different parts of their course in different fibers, as Turner⁹ suggested, and that the axis-cylinders are altered, even when the Marchi degeneration is absent, I am of the same opinion as Turner, that the degeneration of the fibers is sufficient to account for the state of the Betz cells.

The longitudinal section of the spinal cord, stained with thionin showed another interesting picture in some of the fibers of the lateral pyramidal bundles and the cerebellar afferent bundles. As Figure 6 indicates, those fibers show in their course variously long stretches of a deeply blue stain, inserted between colorless parts of the fibers. These blue-stained segments of the fibers hold their stain even if the differentiation by alcohol is pushed further, and to my mind they correspond to the red-stained segments of the fibers in the Alzheimer-Mann preparation described above, and are to be interpreted as degenerated portions of the fibers.

(C) *Manifestations of the Neuroglia Tissue.*—In cases of "central neuritis" an increase of satellite cells as well as of free nuclei was described by Dr. Meyer,¹ Lambert,^{5, 10} Sims¹¹ and others. Our first case showed only moderate increase of satellite cells around the Betz cells and some of the pyramidal cells, but the second case did not give the picture of satellitosis. Lambert¹⁰ observed nests of neuroglia

8. Cole, S. J.: On Changes in the Central Nervous System in the Neuritic Disorders of Chronic Alcoholism, *Brain*, Lond. **25**: 1902.

9. Turner, J.: An Account of the Nerve Cells in Thirty-Three Cases of Insanity, *Brain*, Lond. **26**: 1903.

10. Lambert, C. I.: Demonstrations of Pathological Anatomical Cases, *N. Y. State Hosp. Bull.* **2**: No. 3, 1909.

11. Sims, R.: Anatomical Findings in Two Cases of Korsakow's Symptom-Complex, *J. Nerv. & Ment. Dis.* **32**: 1905.

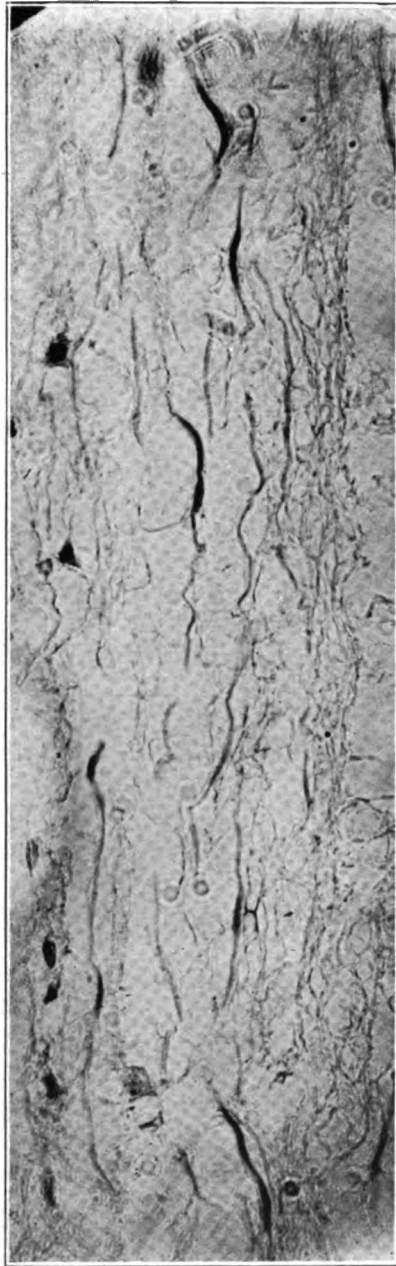


Fig. 5.—Longitudinal section of the cerebellar afferent bundle of the spinal cord (first case).

cells, in which from twenty-five to fifty were closely crowded together, and he said that they multiply by direct division. Similar nests of from three to fifteen neuroglia cells were found in both of our cases (Fig. 8a). As Lambert¹⁰ suggested, they rarely show mitosis, and I also think they divide in the way of amitosis, partly at least.

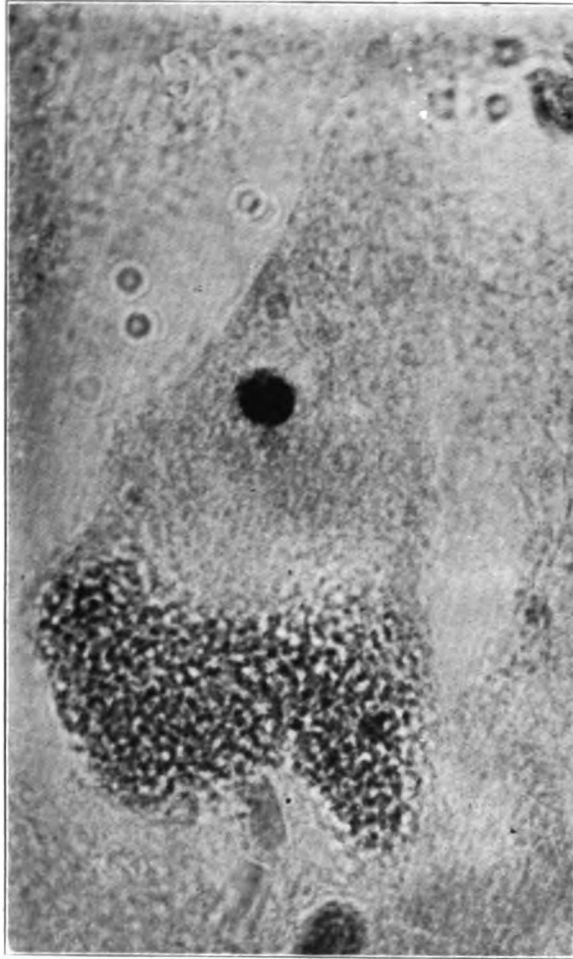


Fig. 6.—A Betz cell after pancreatin digestion.

Special attention was paid to the so-called catabolism process and the catabolism products. In both cases we observed the production of a number of ameboid glia cells, carrying different kinds of granules, which will be described later. Young ameboid glia cells show relatively small amounts of protoplasm, while larger and older ones have

much protoplasm and also protoplasm processes. Keeping pace with the appearance of these ameboid glia cells we find regressive changes of the neuroglia nuclei; these are: homogeneous stain of the nuclei, dilapidation of the nuclei into small spherules and pale stain with evidence of swelling. It is worthy of special note that in our second case we found a large number of neuroglia cells, the nuclei of which are quite small and round and deeply and diffusely blue stained with thionin, and containing a large deeply blue-stained nucleolus (Fig. 8 *a, b*). These nuclei are mostly surrounded by a certain amount of greenish, yellowish pigment, which evidently belongs to the cell body of the neuroglia cells. I think that these nuclei are also in a state of regressive alterations.

Prior to the description of ameboid glia cells, I should like to call attention to an interesting appearance of the neuroglia cells in the marrow of the paracentral lobule. As Figure 8 *d* shows, thionin preparation disclosed many spindle-shaped cells, the long axis of which runs parallel with the nerve fibers in the marrow. The nuclei of these cells are oval or oblongate and deeply blue stained, and have a deeply blue stained large nucleolus; the cell body is also diffusely blue stained or sometimes it shows a honey-comb appearance and tapers off at both ends. From each end of this spindle cell there extends a varicose more or less dark blue-stained fiber. The whole picture gives the impression that the glia cell is lying on an altered nerve fiber. This impression is strengthened, when we study the Mallory, Mann and Alzheimer preparations. In all these preparations we find in the marrow of the paracentral lobule many a spindle-shaped cell with a homogeneously stained oblong or oval nucleus containing a large nucleolus. The relation of the cell and the underlying nerve fiber is clearer in these preparations, because the latter is demonstrated clearly. The cell protoplasm of this spindle cell carries different kinds of granules, which will be described later. It will be noted here that in the spinal cord section of our first case the same spindle-shaped glia cells lying on the nerve fiber were observed in the pyramidal bundle and the cerebellar afferent bundle. I think that this finding is identical with the picture shown in Alzheimer's¹² Text Figure 3 A. Ameboid glia cells were found in both of our cases in the cortex as well as in the marrow, often forming a group independently or in the neighborhood of the blood vessels. Also in the spinal cord of the first case I found a large number of ameboid glia cells.

12. Alzheimer, A.: Beiträge zur Kenntnis der pathologischen Neuroglia und ihrer Beziehungen zu den Abbauvorgängen im Nervengewebe, *Histolog. u. Histopatholog. Arbeiten* 3, 1910.

The ameboid glia cells with large protoplasm show one or multiple vacuoles of different sizes, which may be well explained as fat cysts; the Herxheimer stain revealed red stained, variably large fat drops around the neuroglia nuclei. The Mallory and the Mann preparations showed in a relatively small number of the ameboid glia cells and the above described spindle-shaped glia cells the Alzheimer methyl blue granules. The Alzheimer fuchsin light green preparations, however, demonstrated in a large number of both kinds of the cells the fuchsinophil granules. The Alzheimer light green granules never did come to our observation; nor did the fibrinoid granules come to observation in our Weigert's neuroglia preparations. Besides the methyl blue and fuchsinophil granules the thionin preparations brought to light in a large number of the ameboid glia cells another kind of granule, to which we wish to devote the next chapter.

THE "NUCLEOPROTEID-LIKE GRANULE" IN THE NEUROGLIA CELLS

The granule referred to was demonstrated in the protoplasm of ameboid glia cells in the marrow and also (a little less) in the cortex. Our second case of "central neuritis" showed much more numerous cells with this granule. Besides the ameboid glia cells with large cell body, we found the same granule also in those glia cells, which have a more or less large cell body, but not the characteristic shape of an ameba (preameboid glia cell Rosenthal¹³). The grouping of the granules is quite different due to the different shapes of the cell body; sometimes we find granules all around the nucleus, and sometimes we find them either on one side or on both sides of the nucleus. Here and there we meet groups of granules without showing the neuroglia nuclei, especially often in the neighborhood of blood vessels; they are evidently sections of cell body or protoplasm processes of the glia cells, carrying the granules; according to my observation the latter never lie freely in the tissue or in the perivascular spaces.

The granule is round, or has the shape of an irregular lump; it is rather small and of almost uniform size. It appears in the thionin preparation of both formalin and alcohol fixed materials in a blue stain or in an exquisite metachromatic color, and between the blue stained and the metachromatic basophil stained ones there appear granules with many transition colors (Fig. 8 a, c). In the illumination by electric light they are especially brilliantly visible, and even the blue-stained granules glisten with a more or less metachromatic tinge.

13. Rosenthal, S.: Experimentelle Studien über amöboide Umwandlung der Neuroglia, Referat. Ztschr. f. d. gesamt. Neurol. u. Psychiat. Referat. u. Ergebnisse 8: 1914.

Moreover, the electric illumination disclosed the same granules even in the spindle-shaped glia cells described above, the protoplasm of which appeared in a diffusely blue stain in the daylight. The whole picture of the granules is thus far similar to the granules which I¹⁴ observed in the ameboid glia cells in the synapse of the Mauthner cell in fatigue. As I was not able at that time to study the solubility of that granule, I identified it with the Alzheimer basophil metachromatic granule, which Alzheimer himself identified with Reich's granule on the basis of its morphologic character. The Reich granule is characterized as soluble in warm alcohol at 45 C., ether and in warm xylol. The question now arises whether our granule is identical with the Reich granule, because our specimen showed the granule despite the fact that it had gone through warm xylol in process of embedding. The present cases furnish sufficient material to investigate the microchemical nature of our granule. The fact that we find the granule in the embedded specimens speaks from the first rather against the probability of its being a fat or lipoid substance. Paraffin sections from 2 to 10 μ thick were placed in alcohol (80 per cent., 95 per cent., and absolute), ether, equal mixture of ether and absolute alcohol, chloroform xylol and turpentine oil, and kept in a water bath at 70 C. for from one to three hours, and then were stained with thionin in the same way as the control sections from the same block. Microscopic examination showed no definite decrease in the amount of granules.

Acetic acid (diluted as well as concentrated), concentrated hydrochloric acid, nitric acid and sulfuric acid were applied to sections for two to three hours; however, they did not affect the characteristic stainability of our granule. On the other hand, our granule is quite easily soluble in diluted as well as concentrated alkaline solution; 0.5 per cent. aqueous solution of sodium hydrate dissolves the granule in a short time.

A very interesting picture for the microchemical characterization of our granule was given by the digestion experiment. Paraffin sections from 2 to 6 μ thick were digested by pepsin hydrochloric acid solution at 37 C. for from twelve to eighteen hours. The ground substance of the protoplasm of ameboid glia cells was dissolved, whereas our granule remained undigested around the nuclei, and much more distinctly visible after the digestion. Moreover, it is interesting to note that the granule showed the metachromatic color much more beautifully after the digestion, and it is noteworthy that the granules,

14. Marui, K.: The Effect of Overactivity on the Morphological Structure of the Synapse, *J. Comp. Neurol.* **31**: 1919.

which appeared in a more or less blue tinge before the digestion, showed an exquisite metachromatic color after the experiment.

Extremely interesting is the fact that in the experiments thus far described our granules behave microhistochemically in quite the same manner as the Nissl bodies of the nerve cells; as Held¹⁵ described in 1895, the Nissl substance was dissolved only in alkaline solution as in our experiments, but not in different kinds of acid, in fat-solving mediums, and by pepsin. The only difference consists in the fact that our granules are demonstrated in a more or less brilliant metachromatic color, whereas the Nissl bodies appear in a more blue violet color.

Now, as Held¹⁵ observed, the Nissl bodies show an exquisitely granular structure, if we look at very thin sections (2μ thick). The granules are round and do not look much larger than our granules in the neuroglia cells, and show a more or less metachromatic tinge, especially in electric illumination. After pepsin digestion they appear in a very characteristic metachromatic color in the thionin stain, just as some of our granules in the neuroglia cells. I cannot give a definite explanation to this interesting phenomenon; the fact does, however, indicate further evidence that both granules resemble each other. For the further characterization of our granules I undertook pancreatin digestion of sections. Paraffin sections, from 2 to 6μ thick, were placed in a neutral pancreatin solution for several hours at 38 C., and then they were stained in the ordinary way. Contrary to the result of pepsin digestion the granules of the neuroglia cells in the cortex as well as in the marrow disappeared entirely. The cortex gave a peculiar picture after this procedure; as Figure 6 shows, the Nissl bodies are entirely gone, and we can locate the nerve cells only by the existence of the yellowish pigment and also by the appearance of the nuclei. After five hours' digestion the nuclei of the nerve cells and the glia cells were stained rather distinctly and the nucleoli of the nerve cells were stained pale. Hence, in this point too, our granule reacts microchemically in the same way as the Nissl substance. The microhistochemical reaction of phosphoric acid was done following the direction of Lilienfeld and Monti;¹⁶ our granule and the Nissl substance showed a slightly positive reaction, as they appeared in a brownish, yellowish color.

Judging from all the results thus far described, I came to the conclusion that our granule in the neuroglia cells has chemically a close relation to the Nissl substance. Since the thorough microchemical

15. Held, H.: Beiträge zur Struktur der Nervenzellen und ihrer Fortsätze. Arch. f. Anat. u. Physiol. Anat., Abt., 1895.

16. Lilienfeld and Monti: Ueber die mikrochemische Lokalisation des phosphors in den Geweben, Ztschr. f. physiol. Chem. 17: 1893.

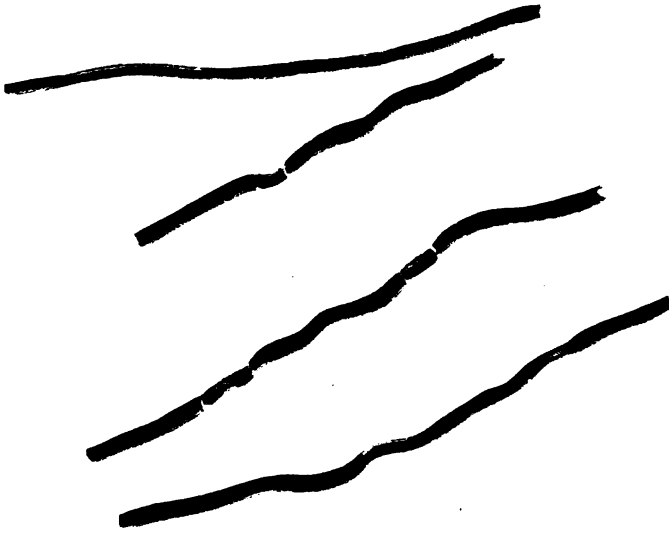


Fig. 7.—Marrow of the anterior central convolution (second case); Alzheimer-Mann's stain.

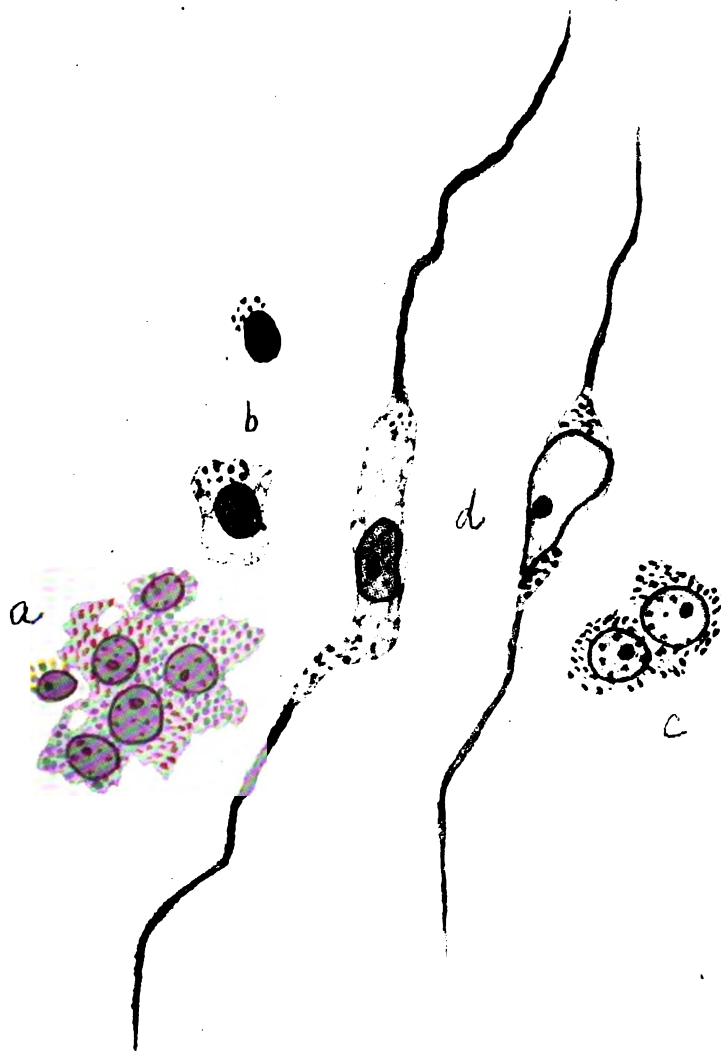


Fig. 8.—Different manifestations of neuroglia cells in "central neuritis" cases.



Fig. 9.—Granule cells and blood vessels in a small fresh hemorrhage.

study of Held on the Nissl substance there is no doubt that the latter belongs to the category of nucleoproteid; so I would like to designate our granule as "nucleoproteid-like granule" in the neuroglia cell. There is no doubt then that our granule is chemically quite different from the Reich π -granule; I therefore made a mistake when I identified the granule in the ameboid glia cells found in the synapse of the Mauthner cell in fatigue with the Alzheimer basophil metachromatic granule (Reich's π -granule). In what relation our granule stands with the so-called "albumin-like granule," which was found in the granule cells in the brains of new-borns, and on which there was a controversy between Virchow^{17, 18} and Jastrowitz,¹⁹ I do not know. As far as I can see, the only fact known about that granule is that it is soluble in alkaline solution; a further study is required to decide this question. It is also doubtful now whether the Alzheimer basophil metachromatic granule is really identical with the Reich granule. He described his granule as stained in a metachromatic color, and as especially visible in electric illumination; but these properties are not specific for the Alzheimer granule nor for the Reich granule, as our granule has the same qualities. Alzheimer admitted, moreover, that the morphology of his granule is somewhat different from that of the Reich granule, and also that the solubility condition appeared to him a little different. He stated that his granule decreased distinctly by putting the section in alcohol. So this question also must be reserved for further investigation. At least, we can state that a metachromatic stain and electric illumination do not by any means suffice for the characterization of his granule.

Under the heading of "simple basophil catabolism substances," Alzheimer¹² described several basophil substances, which show no, or very little, tendency to metachromatic stain. His description is somewhat vague. He characterized one of them through the fact that (1) it is demonstrated only in alcohol sections, not in formalin material; (2) it is found in nerve cells, in neuroglia cells and also in groups in the perivascular spaces, and (3) it is demonstrated much more in the cortex than in the marrow. This granule is quite different from our granule in many respects. Another kind of basophil products of Alzheimer (Table 33, Fig. 3) is found, according to him, in the neuroglia cells, especially in the marrow, and demonstrated in the shape of lumps. To my mind, this granule resembles somewhat our

17. Virchow, R.: *Encephalitis Congenita*, Berl. klin. Wchnschr., No. 46, 1883.

18. Virchow, R.: *Verhandlungen d. Berlin med. Gesellschaft. Sitzung. v. 14 Oktober, 1883*, Berl. klin. Wchnschr., No. 46, 1883.

19. Jastrowitz: *Diskussion zu den Vorträgen von Jacusiel u. Virchow in den Sitzungen der medizinischen Gesellschaft*, Berl. klin. Wchnschr., No. 46, 1883.

granule, but I am far from being able to say that they are both the same thing. As already remarked, sometimes our granule is demonstrated in a basophil stain, showing little tendency to metachromasia. So I must declare that whenever we speak of the latter kind of the Alzheimer "basophil catabolism product," we have to exclude first in a microchemical way our "nucleoproteid-like" granule. As far as I know, our granule is quite new, at least in so far as its chemical nature is determined.

Then the question comes, whether our granule is connected with any specific morbid condition of the central nervous system and how frequently we meet this kind of granule in the neuroglia cells. Through the kindness of Dr. Meyer, I was given the privilege of studying many specimens in our laboratory; in all, I went over thionin preparations of seventy cases, mostly taken from the paracentral lobule. The cases belonged to both insane and general hospital cases, and the age of the patients ranged from 2 weeks to 68 years. To my surprise, I found the same granule very easily in twenty cases, and with some effort in twenty-five cases and none in the rest. Generally speaking, the granule was demonstrated in typical ameboid glia cells or in glia cells, the protoplasm of which is more or less larger than usual (preameboid glia cells Rosenthal?), in the cortex as well as in the marrow. So far as my observation went, I could not make out any specific cause for the appearance of the granule. As a most interesting finding I emphasize here that in a case of aplastic anemia, I found in and around a small fresh hemorrhage in the corpus callosum a large number of granule cells loaded fully with the "nucleoproteid-like granules." Newly produced granule cells have round or more or less simple shapes, and do not show any basket-like structure of their cell body; the latter is filled with the granules. Those young granule cells, which have a more or less irregular shape, have a close resemblance with ameboid glia cells (Fig. 9, *a*). On the other hand, the granule cells show an exquisite tendency to form basket-like structures, and I observed all the transition forms between the young granule cells and the typical basket-like granule cells; in other words, we found many cells with both basket-like structure and a more or less large protoplasm rest. Extremely interesting was the finding of the nucleoproteid-like granules in this protoplasm rest and in the ribs between the basket meshes (Fig. 9, *a*, *b*, *c*). There were also found many phagocytic cells in the hemorrhage, which contained many red blood corpuscles in their cell body. Especially noteworthy was here as well the finding of the same granules between those red blood corpuscles. These findings are very interesting in so far as a new chemically well-defined granules were observed in granule cells. The

question whether these granule cells have something to do with those granule cells in the brains of new-borns is to be decided by further investigation. The cells of blood vessels show in and around this hemorrhage very distinct progressive manifestations. We observed new production of cells of blood vessels, which show a tendency to form mesodermal granule cells and also to form new capillaries. These cells all carry in their protoplasm the same "nucleoproteid-like granules" (Fig. 9 d). In two cases of "central neuritis" we observed the granules only here and there in the protoplasm of swollen adventitia cells of blood vessels, around which many ameboid cells with different granules were found crowded. Herxheimer's stain showed variously large fat drops in the ameboid glia cells, as well as in cells of the adventitia. The question, whether the "nucleoproteid-like granule" appears only in certain morbid conditions of the central nervous tissue, or whether it is a normal constituent of neuroglia cells, which is found increased in quantity in morbid conditions, I cannot answer definitely at present. Although I found this granule in brains, which showed no definite histopathologic manifestation, except for the fact that the neuroglia cells had a more or less larger protoplasm body, we can never be sure in our necropsy material that we are dealing with absolutely normal brains. It would be necessary to add here that in two "normal" sheep brains I did not observe any neuroglia cell with this granule.

BIOLOGIC SIGNIFICANCE OF THE "NUCLEOPROTEID-LIKE GRANULE" IN THE NEUROGLIA CELLS

Since the thorough study of Alzheimer^{12, 20} on the pathologic neuroglia, we know that in certain pathologic conditions the neuroglia tissue produces ameboid glia cells, which work as scavengers of catabolism products of the central nervous tissue. Alzheimer¹² described several catabolism products with different morphologic character and staining reaction. His methyl blue granule is produced according to him from the protoplasm of cells, which are bound to rapid decay, and dissolved and transformed into fat substance by mesodermal cells and carried away. Rosenthal,¹⁸ who wanted to explain the production of the methyl blue granule as a sign of necrobiosis of neuroglia tissue, regarded the appearance of the fuchsinophil granule as that of an increased scavenger activity. So there is no doubt, that in our cases of "central neuritis" ameboid glia cells are

20. Alzheimer, A.: Ueber die Abbauvorgänge im Nervensystem. Referat auf d. VII Jahresversammlung der Gesellschaft Deut. Nervenärzte un Breslau, Sept., 1913, Deutsch. Ztschr. f. Nerven. 50: 1914.

working as scavenger of catabolism products. The "simple basophil catabolism product" was interpreted by Alzheimer as a manifestation of a characteristic dissolution process of cell protoplasm. The so-called basophil metachromatic granule has, according to Alzheimer, something to do with myelin, although he was in doubt whether the granule comes from **decay or nutrition disturbance of myelin fibers**. But the biologic significance of these granules is to my mind to be considered after their nature becomes clearer by further investigation. What then is the biologic significance of our "nucleoproteid-like granule"? Could it also be attributed to catabolism products in the same sense as that of Alzheimer? This is by no means an easy question to answer definitely. That our granule has microchemically a close resemblance to the Nissl granule, I described conclusively above. Now many investigations indicate a close relation between the Nissl substance and nuclear substance with regard to their histological character. Holmgren,²¹ Sjöval²² and I¹⁴ also expressed the ~~opinion~~ **that on the occasion of tigrolysis the Nissl substance is regenerated from the nucleus**; according to Scott, the Nissl substance comes out of the nucleus during the development of the nerve cell. On the other hand, it is known that in reparative activity of root ganglion cells (after section of the nerve cell process) and in specific nervous activity (fatigue) we always observe a change of the chromophil substance. Heidenhain²³ said that this substance is accumulated in the rest state, whereas in activity it undergoes specific metamorphosis and produces specific energy. It is then highly improbable that our granule, which is chemically closely related to such an important substance, should be given to ameboid glia cells and other cells as a catabolism product of the nervous tissue. Also the fact that our granule is found in larger quantity in the marrow than in the cortex, speaks rather against this view. The postmortem appearance of ameboid and preameboid glia cells was studied by Buscaino,²⁴ Rosenthal¹³ and Wohlwill,²⁵ but it is hard to conceive a postmortem production of our granule. Then, we must appeal to another possibility — whether our granule is given to neuroglia cells in an afferent

21. Holmgren, E.: Studien in der feineren Anatomie der Nervenzellen, Anatomische Hefte **15**: 1900.

22. Sjövall, E.: Die Nervenzellenveränderungen bei Tetanus und ihre Bedeutung, Jahrbuch. f. Psychiat. u. Neurol. **23**: 1903.

23. Heidenhain, M.: Plasma und Zelle, I, 1911.

24. Buscaino, V. M.: Sulle genesi e Sul significato delle cellule amoeboidei. Referat. Ztschr. f. d. gesamt. Neurol. u. Psychiat. Referate u. Ergebnisse, **8**: 1914.

25. Wohlwill: Ueber amoeboide Glia, Virchow's Arch. **216**: 1914.

direction; in other words, from blood, and is eventually related to a reparative or reconstructive and also nutritive process of the nerve tissue. The finding of the same granule in cells of blood vessels may afford a point of support to this assumption, although we do not know in what chemical form it is given and in what chemical process it is produced in the cells.

Extremely interesting is the finding of the "nucleoproteid-like granule" in granule cells. Merzbacher²⁶ studied the morphology and biology of granule cells, to which he gave the designation "Abräumzellen"; among the granule cells, which are found in embryonic brains, he distinguished two categories: "physiological granule cell" ("embryologische Auffauzellen") and "Abräumzellen" in a narrower sense ("granulare metamorphosierte Gliazellen"). He said in his conclusion: "There is no decided difference between the 'Abräumzellen' and 'Auffauzellen'; they both have the property to take material and work it out and to pass it on. The distinction between the two comes into consideration when we follow the fate of the material, which was given to the cells. In the one case it is carried away, and at another time it is used for the production of a new substance and structure. The study of Wlassak²⁷ on embryonic material strongly suggests the activity of neuroglia during the evolution of the nerve tissue. De Montet²⁸ regarded the myelin as the expression of import and construction as well as of catabolism process. It is, then, perhaps not going too far to assume that in certain pathologic conditions and nutrition disturbances of nerve tissue other chemical substances besides fat substance may be given to the latter by the neuroglia tissue. Moreover, it is easy to understand that the nerve tissue needs some protein substance besides fat as its constructive and nutritive substance. To my mind our granule belongs to this kind of substance. The further question, whether this substance is given as such or if it is also given, after it is changed into some other substances, I shall leave undecided. According to Wlassak the origin of myelin is to be sought in the blood. While fat production from protein substance is decidedly denied by several physiologic chemists (Schenck, Gürber), others (Kunze) spoke for this possibility up to quite recently. Arnold

26. Merzbacher, L.: Untersuchungen über die Morphologie u. Biologie der Abräumzellen im Zentralnervensystem, Histolog. u. Histopatholog. Arbeiten 3: 1909.

27. Wlassak: Die Herkunft des Myelins, Arch. f. Entwicklungsmechn. d. Organ. 4: 1898.

28. De Montet: Ueber Wanderungen lipoider Substanzen im Centralnervensystem, Inaugural Dissertation z. Erlangung d. med. Doktorwürde d. hohen med. tak. d. Universität Bern, 1906.

and Dietrich considered that in certain pathologic conditions fat production from protein substance is possible but not proved. Jakob²⁹ also admitted the possibility of production of lipid catabolism products from axis-cylinders, which go to decay. Our findings also seem to indicate the same possibility, but we cannot consider this is proved. As already remarked, we found in granule cells and ameboid glia cells fat cysts beside the "nucleoproteid-like granule"; but it is always possible that they give one substance on the one hand and take the other on the other hand. In some glia cells we also found yellowish, greenish pigment beside the granule; but I do not know whether this pigment was present before or newly produced, because Obersteiner^{30, 31} showed the existence of this pigment in neuroglia cells of normal brains increasing in quantity with age. Marinesco and Sander assumed the production of pigment of nerve cells from the Nissl substance; Obersteiner regarded it as a residuum of metabolism processes of the nerve cell. In what relation our finding stands with the assumption of these authors I do not know.

So I came to the conclusion that the neuroglia tissue not only has the function of scavenger through production of granule cells and ameboid glia cells in certain pathologic conditions and nutrition disturbances, but also the function of "Auffau" in the sense of Merzbacher.

SUMMARY

The results of a thorough histopathologic study in two cases of "central neuritis" and many other cases can be summarized as follows:

1. Almost all the Betz cells in both cases and some cells of the spinal cord of the first case showed the typical axonal reaction; in the first case the fever alteration was superimposed upon this picture.
2. Fragmentation of the intracellular neurofibrils was found in the glassy area; the alteration of neurofibrils keeps pace with the dissolution of the Nissl bodies.
3. Besides Marchi degeneration of myelin sheaths, a very interesting picture of degenerated axis-cylinders was disclosed.
4. Ameboid glia cells showed the Alzheimer fuchsinophil granule, a finding which indicates an increased scavenger activity of the neuroglia tissue.

29. Jakob, A.: Ueber die feinere Histologie der sekundären Faserdegeneration in der weissen Substanz des Rückenmarks (mit besonderer Berücksichtigung der Abbauvorgänge), *Histolog. u. Histopatholog. Arbeiten* 5, 1912.

30. Obersteiner, H.: Ueber das hellgelbe pigment in den Nervenzellen und das Vorkommen weiterer fettähnlicher Körper im Centralnervensystem, *Arbeiten a. d. Neurol. Institut.* 10: 1903.

31. Obersteiner, H.: Weitere Bemerkungen über die Fettpigmentkörnchen im Centralnervensystem, *Arbeiten a. d. Neurol. Institut.* 11: 1904.

5. In two cases of central neuritis and many other cases in ameboid and preameboid glia cells on the one hand, and in a case of hemorrhage in granule cells on the other hand, a new "nucleoproteid-like granule" was demonstrated and a conclusion was drawn that neuroglia has a constructive function besides a scavenger function, and that this granule is given the neuroglia cells in an afferent direction.³²

32. In addition to the references already given, the following will be found of interest:

Reich, F.: Ueber eine Granulation in den Nervenzellen, *Arch. f. Anat. u. Physiol., Physiolog. Abt.*, 1903.

Reich, F.: Ueber die feinere Struktur der peripheren Nerven, *Ztschr. f. Psychiat.* **62**: 1905.

Reich, F.: Ueber den zelligen Aufbau der Nervenfasern auf Grund mikrohistiochemischer Untersuchungen, *J. f. Psychol. u. Neurol.* **8**: 1906-1907.

Somers, E. M.: Central Neuritis, *New York State Hosp. Bull.* **1**: No. 3, 1908.

A CONTRIBUTION TO THE HISTOPATHOLOGY OF EPIDEMIC ("LETHARGIC") ENCEPHALITIS *

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The various symptoms of epidemic ("lethargic") encephalitis—somnolence, stupor with or without slight elevation of temperature, together with ocular and other cranial nerve paralyses, etc., may occur in many cerebral disorders. We may mention the hemorrhagic superior polioencephalitis of Wernicke, the cerebral form of poliomyelitis, the sleeping sickness caused by trypanosomiasis, syphilitic meningitis, parietic dementia, encephalitis caused by anthrax (Fulci¹) or other infectious diseases, as tuberculosis, cerebrospinal meningitis and especially influenza. Here also belong cases of poisoning with carbon monoxid, sulphuric acid (Wernicke²) and meat and fish (botulism). Any of the types of encephalitis mentioned may so resemble the clinical picture and course of the epidemic ("lethargic") form that they, undoubtedly, have been and are confounded with the latter. Especially striking is the clinical similarity between lethargic encephalitis and the sleeping sickness caused in man by the *Trypanosoma gambiense*, hence "lethargic" encephalitis is popularly called "sleeping sickness." In addition, from our histopathologic studies of three cases of so-called lethargic encephalitis we are forced to the conclusion that the latter, though a disease *sui generis*, does not differ histologically from the African sleeping sickness due to trypanosome infection.

Of the three cases here described, two have been briefly reported by one of us (Bassoe³) and a third case, in a baby 4 weeks old, came under observation of Drs. C. G. Grulee and Peter Bassoe not long ago.

* From the laboratories of pathology of Cook County and Psychopathic Hospitals, Chicago.

* Read before the Section on Nervous and Mental Diseases at the Seventieth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1919.

1. Fulci, F.: Die acute hämorrhagische Leptomeningo-enzephalitis bei der Milzbrandinfektion des Menschen, Nissl-Alzheimer's Arbeiten 6:161, 1913.

2. Wernicke, C.: Lehrbuch der Gehirnkrankheiten 2:229, 1881, Berlin.

3. Bassoe, Peter: Epidemic Encephalitis, J. A. M. A. 72:971 (April 5) 1919.

REPORT OF THE THIRD CASE

History.—A female child, 4 weeks old, vomited frequently for two days, and then repeated attacks of contractions of the diaphragm set in. When admitted to the Presbyterian Hospital, March 14, 1919, she was stuporous, with temperature ranging from 98 to 102 F. and a leukocyte count of 9,950. A laparotomy was performed and appendicostomy made. The following day the temperature rose to 103.2 F. and subsequently generally ranged between 100 and 102. A few days later nystagmus and strabismus were observed. The child died on March 25.

Necropsy.—This was held by Dr. B. O. Raulston and the following anatomic diagnosis was made: moderate hyperemia and edema of the brain; hyperemia of lungs; distention of urinary bladder; bilateral hydronephrosis; moderate dilatation of ureters; recent unhealed laparotomy wound; open stump of appendix; fatty changes in liver; patent ductus arteriosus and foramen ovale.

MACROSCOPIC EXAMINATION OF CASE 3

Macroscopically, the brain appeared practically normal. The pia was somewhat opaque, especially around the cerebellum; it was congested, rusty looking, but easily detachable from the brain substance. The convolutions were flattened, the sulci not gaping, the ventricles slightly dilated, the gray matter distinct from the white substance. There were no hemorrhages, no signs of ependymal changes.

MICROSCOPIC EXAMINATION OF THE THREE CASES

The brains from these three cases and the spinal cord of the third case were delivered to the laboratory fixed in a 10 per cent. formalin solution. Portions from every region of the hemispheres and brain stem, as well as from the spinal cord, were placed in 96 per cent. alcohol (for celloidin sections); some were prepared for paraffin, and a great number of frozen sections were also made from almost every portion of the central nervous system. The sections were stained by various methods, such as Mallory-Jacob, Alzheimer-Mann, Herxheimer (scarlet red), Bielschowsky, Weigert's elastic stain, toluidin blue, thionin, hematoxylin-eosin and many others.

The pia appeared infiltrated and frequently showed distended meshes containing congested, hyperemic vessels. The infiltration cells were mainly lymphocytes and plasma cells, about equally represented and especially numerous around the vessels. Numerous scattered cells were also seen in the meshes of the pial connective tissues (Fig. 1). Both the lymphocytes and the plasma cells appeared very densely stained, the latter showing a distinct cytoplasm and an excentrically located nucleus which exhibited the typical wheel-like arrangement of its chromatin and a distinct membrane. In contrast to these cells, the lymphocytes were represented by darkly stained, often homogeneous, nuclei, without any traces of cytoplasm. The latter was also absent in

many plasma cells which on superficial examination resembled lymphocytes. The plasma cells were not always of a typical appearance, but they were very numerous. Besides the lymphocytes and plasma cells, there were other forms present, but much less numerous, namely, fibroblasts, polyblasts, macrophages and rod cells — *Stäbchenzellen* (Fig. 2). The fibroblasts usually appeared as large, oval or round nuclei, filled with numerous small particles of chromatin, surrounded

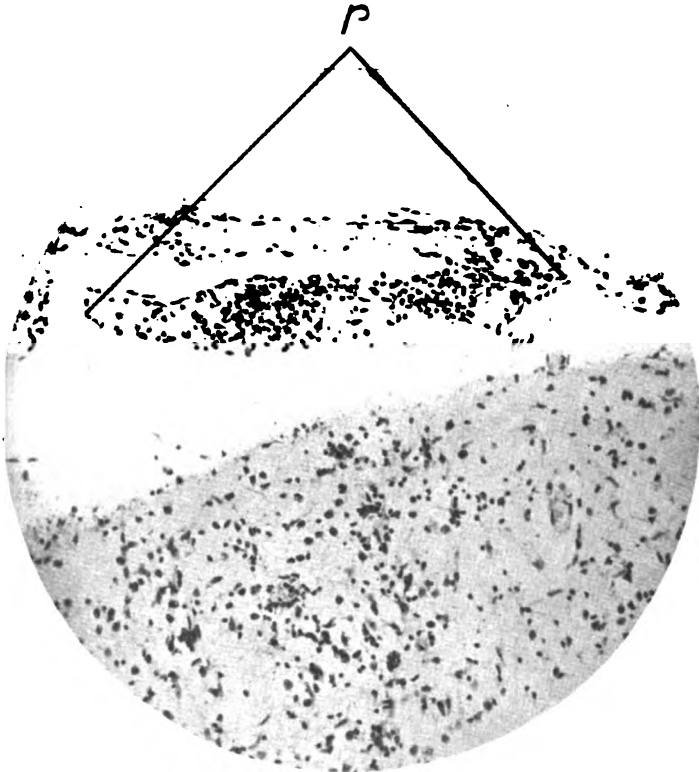


Fig. 1.—Infiltration of pia and cerebellar cortex. *P*, pia infiltrated and separated from the cerebellum. The molecular layer of the latter is much infiltrated by various cells as explained in the text. (Toluidin blue stain, $\times 150$.)

by a well-formed membrane, but without any visible cytoplasm. They could easily be distinguished from the plasma cells and lymphocytes by their larger size and pale nuclei. Still fewer in number were certain other cells also with pale, irregular, often horseshoe-shaped nuclei, enclosed within a pale cytoplasm. These cells—polyblasts—were scattered singly or mixed with the three forms mentioned above.

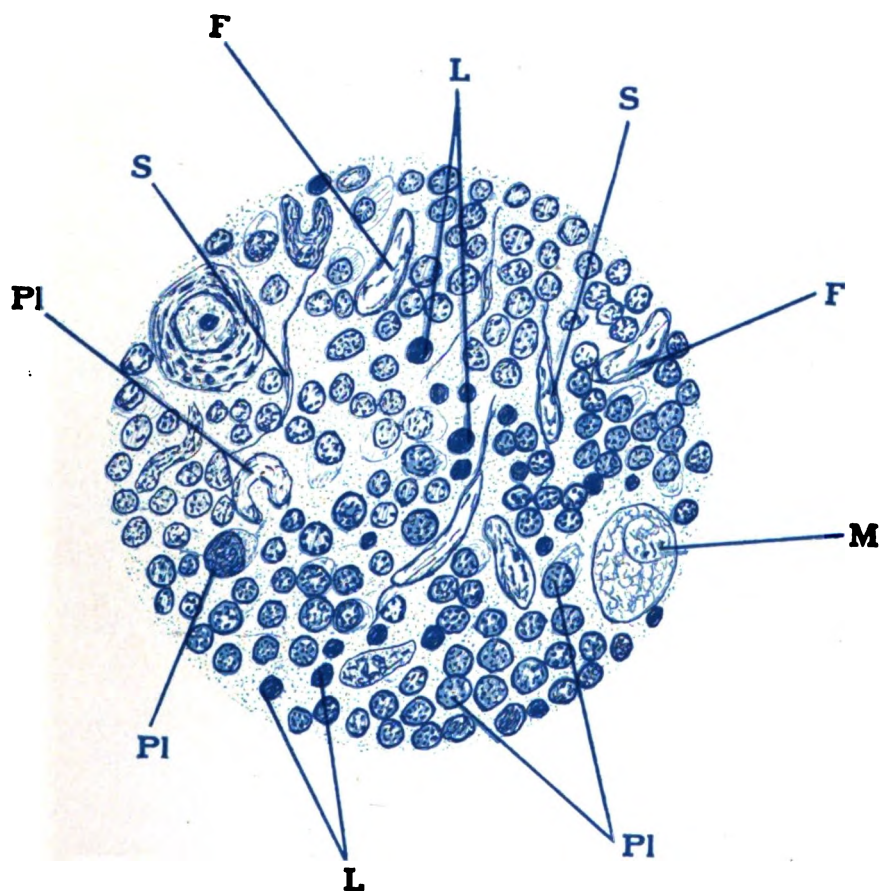


Fig. 2.—Types of various infiltration cells: L, lymphocytes; Pl, plasma cells; M, macrophage; Pb, polyblast; F, fibroblasts; S, *Stabchenzellen*. Toluidin-blue stain.

The macrophages were exceedingly rare, often contained small particles of blood pigment, or distinct vacuoles, and an excentrically located nucleus. The macrophages were seen only in those places where blood pigment was present, mostly as very minute particles.

Toluidin-blue specimens frequently revealed a great number of rod cells (*Stäbchenzellen*) especially near the vessels. They exhibited an

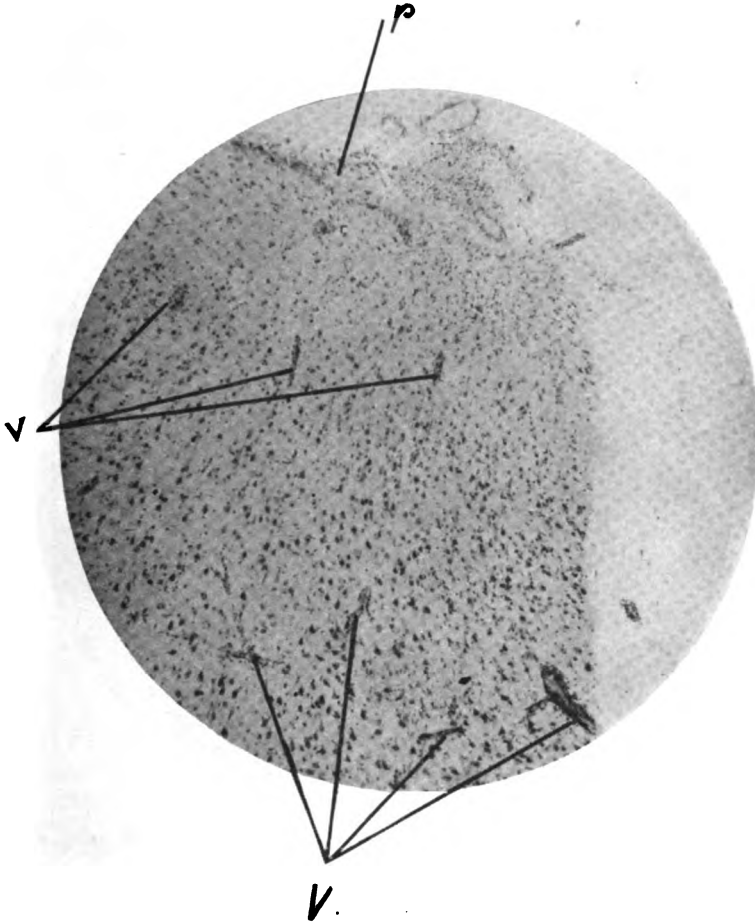


Fig. 3.—Cortex from the Island of Reil; the vascular infiltration especially in the deeper strata is very marked; the pia (*P*) is also infiltrated, but slightly. *V*, vessels. (Toluidin-blue stain, $\times 50$.)

oblong, distinctly stained nucleus, with chromatin dots, and very pale cytoplasmic processes, emanating from the opposite poles. Any other cellular elements, such as mast cells, gutter cells and polymorphonuclear cells were not found. The infiltration was in evidence all over the pia, that is, around every part of the brain, but was especially marked

around the cerebellum, more so at the base, and it frequently involved the adjacent parenchyma. Figure 1 distinctly shows the invasion by the infiltration cells of the molecular layer of the cerebellum.

CLINICAL SIGNIFICANCE OF THE CHANGES OBSERVED

The pial changes, as outlined, indicate a mild leptomeningitis, more marked in some portions than in others, but not so pronounced as in paralytic dementia or in poliomyelitis. However, the pial changes in their character and localization, though less intense, very much resemble those described and pictured by Mott,⁴ Spielmeyer and others⁵ in



Fig. 4.—Pial vessels. The elastic membrane in the form of solid rings is very clear. (Resorcin-fuchsin stain, $\times 60$.)

4. Mott, F. W.: The Changes in the Central Nervous Systems of Two Cases of Negro Lethargy, *Brit. M. J.* **2**:1666, 1889. The Cerebrospinal Fluid in Relation to Diseases of the Nervous System, *Brit. M. J.* **2**: (Dec. 10) 1904. The Examination of the Tissues of a Case of Sleeping Sickness in a European, *Brit. M. J.* **1**: 1904. Histologic Observations on the Changes in the Nervous System in Trypanosome Infections, Especially Sleeping Sickness, etc., *Arch. Neurol.* **3**:581, 1907.

5. Franca and Athias: Les Plasmazellen dans les vaisseaux de l'écorce cérébrale, dans la paralysie générale et la maladies du sommeil, *Compt. rend. soc. de biol.* **54**:192, 1902. Spielmeyer, W.: Die Trypanosomenkrankheiten, Jena, 1908; also, Schlafkrankheit und Progressive Paralyse, *München. med. Wchnschr.* **54**:1065, 1907.

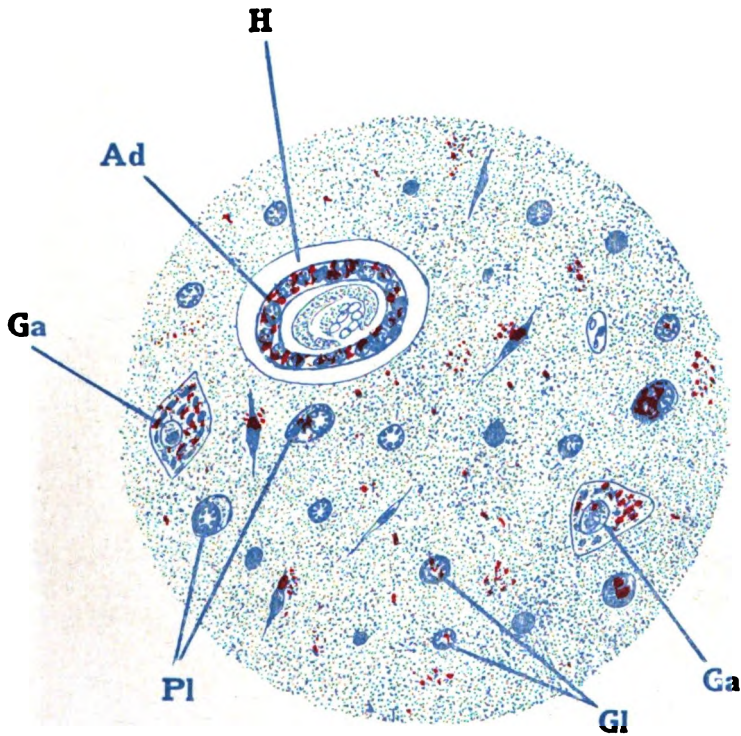


Fig. 5.—Substantia nigra. The ganglion cells, the glia nuclei, the adventitial space show fat globules which are also scattered all over the glia network in the form of minute droplets (Hematoxylin and Scarlet-red stain of Herxheimer.) *Gl*, glia nuclei; *Pl*, plasma cells; *Ga*, ganglion cells; *H*, His' space (Nissl's *Schrumpfraum*); *Ad*, adventitial space of Virchow-Robin.

trypanosomiasis. Vascular changes are much less constant and much less pronounced in the cortex of the cerebrum and the cerebellum. Dozens of sections may not show a single markedly infiltrated vessel. The few cortical perivascular infiltrations seen are small and, like the pial ones, more numerous in the cerebellum. Nevertheless, the cerebral cortex shows an abundance of other pathologic changes. The architecture of the various cell layers is practically unaltered, though some regions such as the occipital lobe, do not appear as richly supplied with ganglion cells as normally.

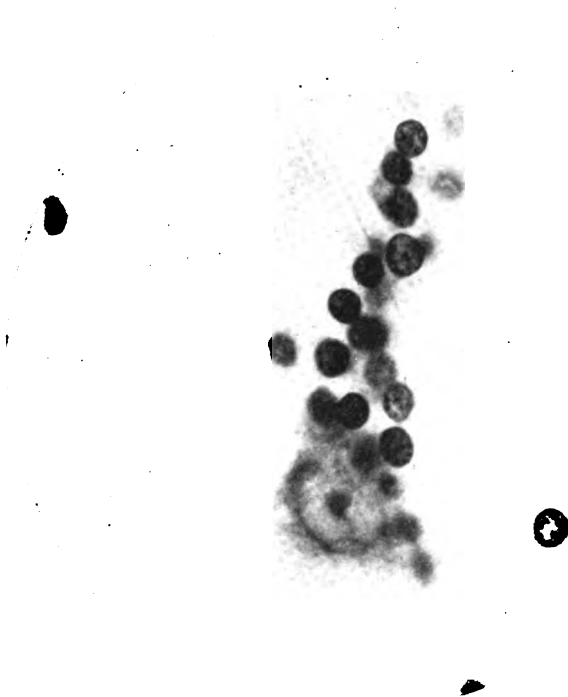


Fig. 6.—Type of neurophagia in the cortical and subcortical regions. The apical process as well as the right basal one are densely covered by glia cells. The apical process is still visible beneath the glia cells. The cell body is very pale, the chromatin accumulated as a perinuclear cap. The round, free bodies scattered over the picture are also glia cells. (Toluidin blue stain, $\times 100$.)

CHANGES IN THE CORTICAL LAYERS

As a rule, the cortical layers contain a great number of glia cells, mostly in the form of so-called glia nuclei, as well as an abundance of capillaries. Some sections exhibit a rich capillary network, the vessels appearing much congested, but without any signs of sprouting, endothelial proliferation or perivascular infiltration. Their intima shows a

well-stained endothelium, a free lumen not obscured or narrowed by a proliferated intima. The elastic membrane is homogeneous, not split, powerfully developed and solidly blended with the intima. All these features are seen in Figure 4, which is taken from the pia whose vessels are identical in appearance with those of the brain. Many capillaries, in Herxheimer (scarlet red) specimens, show minute and larger drops of fat enclosed within the adventitial cells, in some instances forming a solid ring around the lumen (Fig. 5).

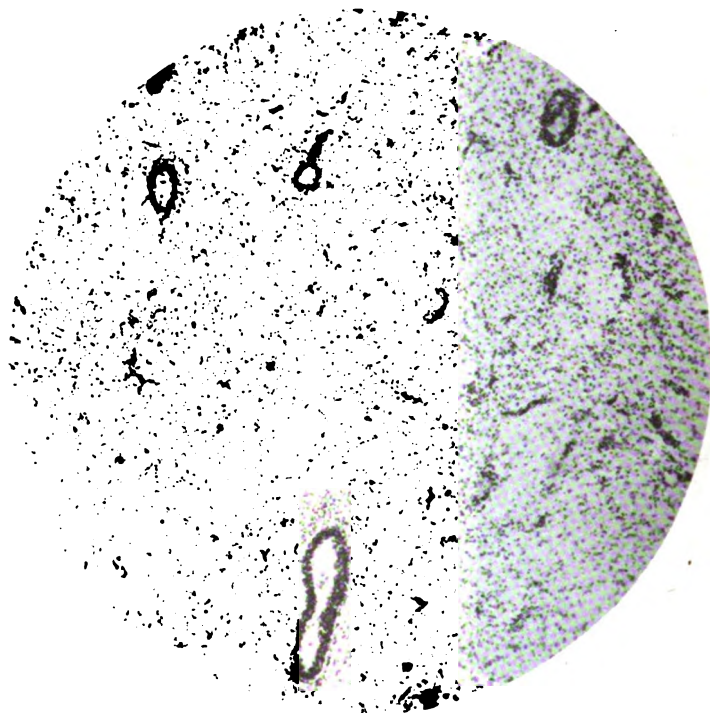


Fig. 7.—Infiltration of the optic thalamus, with numerous elements, explained in text. As compared with Fig. 5 the thalamus seems to be even more affected than the substantia nigra. (Toluidin blue stain, $\times 60$.)

Generally speaking, the cortical mesodermal elements show few pathologic changes. These are, however, quite pronounced in the ectodermal elements, namely, the glia and the ganglion cells. Some of the latter are of normal size and structure, but the majority appear rather homogeneous, uniformly stained, without Nissl bodies, and with a dark, pyknotic or very pale, granular nucleus. In some cells the disintegration goes so far that only the nucleolus is visible. The cell processes are mostly densely stained, homogeneous, tortuous and without any traces of Nissl substance. All types of cell changes described by

Nissl are encountered, from the milder types, so-called chromatolysis and acute cell disease type, to the severe type when the cell body, including the nucleus, is reduced to an amorphous, granular mass invaded by glia cells—"neuronophages" (Fig. 6). Other less damaged cells are merely thickly surrounded by them (satellitosis). Satellitosis, chromatolysis and neuronophagia are the most common alterations to be met with in the cortex, especially in the deeper strata. In scarlet red specimens many cells, especially in the deeper layers, show droplets of fat

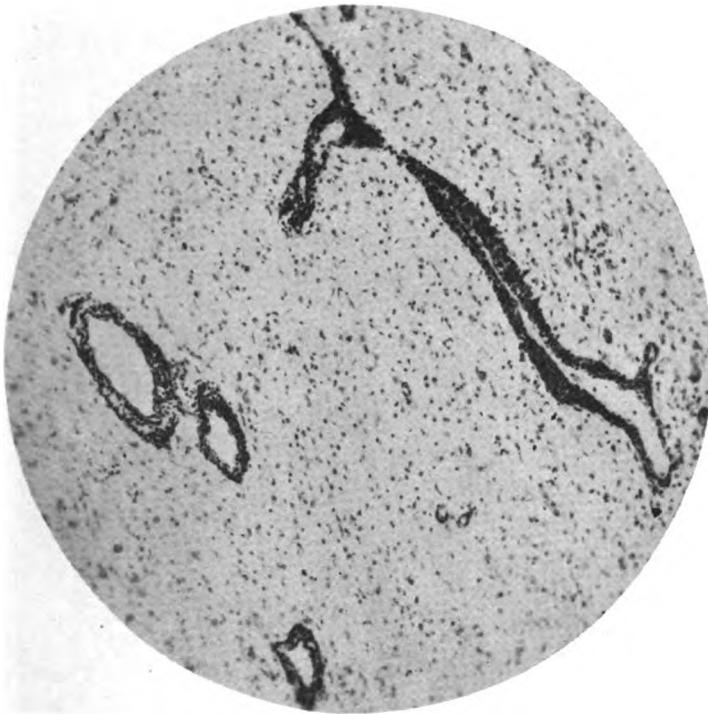


Fig. 8.—Substantia nigra is covered by densely infiltrated vessels and numerous glia nuclei, especially to the right of the larger vessel. Some of the dots are free plasma cells and other bodies, as explained in text. (Toluidin-blue stain, $\times 110$.)

mostly at the base of the cell, often surrounding the nucleus in the shape of a semicircle. The glia tissue, as we said, shows a marked proliferation of its cells, some of which have abundant protoplasm, but the majority are in the form of large nuclei rich in chromatin (Fig. 5). Astrocytes are absent, and glia fiber proliferation, as well as regressive changes, like ameboid glia, "fill bodies" (*Füllkörperchen* of Alzheimer), various granula and glia reticulum are totally lacking.

These parenchymatous changes are much more marked in the subcortical regions, the basal ganglia, subthalamic area and tegmentum. In these regions they are not only very pronounced, but they are also combined with profound interstitial changes. Thus, the vascular infiltrations are enormous (Figs. 7, 8, 9), in the case of some smaller arteries compressing their lumina, giving them the shape of a figure 8, or in the case of capillaries and small veins totally obscuring their shape (Fig. 9). The infiltration elements—lymphocytes, plasma cells, poly-

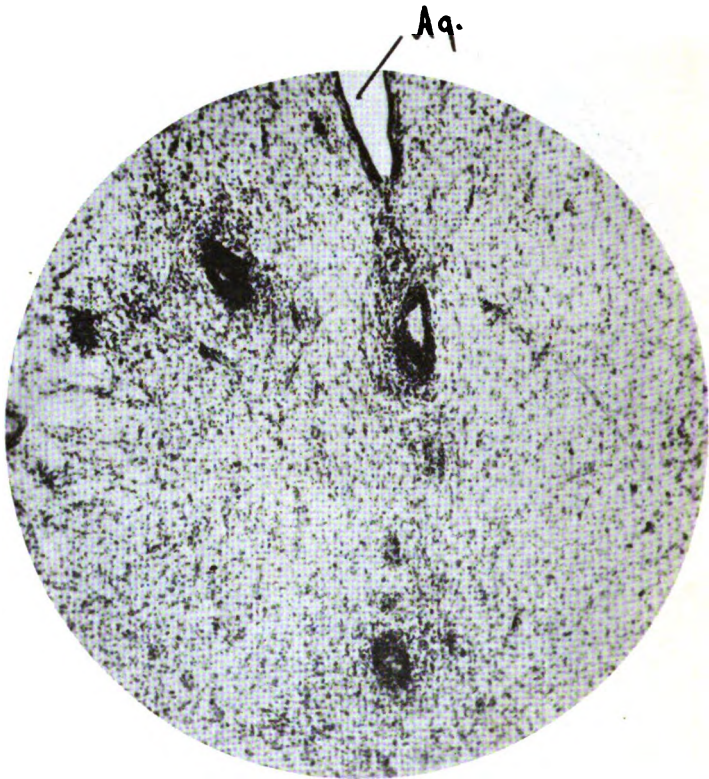


Fig. 9.—Tegmentum of pons. Infiltration of the vessels and of the formatio reticularis. *Aq.*, Aqueducts Sylvii covered with ependyma cells. (Toluidin-blue stain, $\times 65$.)

blasts, fibroblasts, rod cells—are principally confined to the adventitial spaces of Virchow-Robin, but sometimes they are so dense that these spaces seem to be overflowed by the cells, and an impression is gained that the space outside the adventitia, between the latter and the brain—the so-called perivascular space of His—is also invaded. Careful observation, however, shows that the latter is usually free, and that the infiltration is exclusively confined to the Virchow-Robin spaces as well as

to the distended meshes of the adventitia. In some places, like the optic thalamus and substantia nigra, the infiltration cells can be seen invading the parenchyma itself (Figs. 10, 11, 12), yet it is sometimes difficult to make out whether the invading elements (plasma cells and rod cells) traveled from a densely infiltrated vessel or are merely the focal products of inflamed or infiltrated minute capillaries.

CELLULAR ELEMENTS FREELY PRESENT

At any rate, some parts like the optic thalamus and substantia nigra, show a wealth of various cellular elements, including glia cells, all mixed in irregular masses, which surround vessels, capillaries and

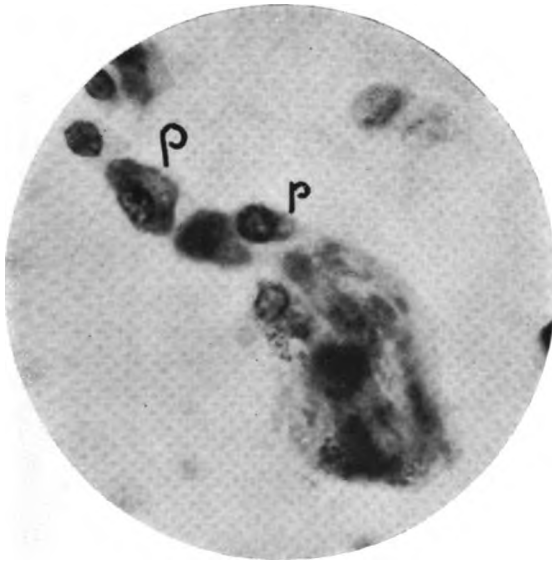


Fig. 10.—Typical plasma cells (*P*) along a dendrite of a deeply changed ganglion cell. (Toluidin-blue stain, $\times 1,200$.)

ganglion cells. Many vessels, especially the smaller ones, are not only densely infiltrated, but also congested, packed with blood pigment, some containing hyaline and other thrombi (Fig. 13). In spite of such marked infiltrative phenomena, some ganglion cells—especially in the substantia nigra—appear quite normal, though the majority show various changes (chromatolysis, neuronophagia and even cell sclerosis). As in the pia and cortex, hemorrhagic foci, gutter cells, foci of softening and polymorphonuclear cells are totally lacking. Only very minute capillary hemorrhages were found, and once a larger hemorrhage could be seen surrounding a congested small vein. In both instances, however, reactive phenomena in the neighborhood of the hemorrhagic foci

were absent, and it is probable that these hemorrhages were terminal or agonal. In this connection, we should mention numerous red bodies—spherules—throughout the spinal cord of Case 3, in the gray as well as the white matter (Fig. 14). They are round and regular in shape, lying singly in the parenchyma or near the capillaries and even in the perivascular, adventitial spaces. They are smaller than red cells, are much more densely stained with eosin, are perfectly homogeneous and not enveloped by glia tissue. The latter is not thickened or proliferated around them. It is hard to tell at the first glance whether these bodies are plain erythrocytes, or formations described as cystic plasma cells (cysto-plasmatozyta) by Perusini⁶ and also depicted by Favorsky⁷ in

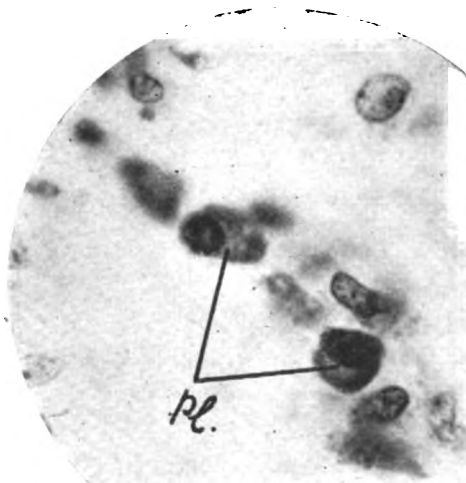


Fig. 11.—A degenerated ganglion cell (from substantia nigra) invaded and surrounded by plasma cells (Pl). (Toluidin-blue stain, $\times 1,200$.)

a case of tabes dorsalis. Still more do they resemble the bodies described by Fulci,¹ so it would appear proper to call them the *spherules of Fulci*. They show very well in Alzheimer-Mann specimens, appear in Bielschowsky specimens as round, pale brown bodies, and do not show at all with thionin, toluidin blue, hematoxylin-eosin or scarlet red stains, while in the brain tissue they cannot be demonstrated at all.

6. Perusini, G.: Sopra Speciali Cellule Degli Infiltrati Nel Systema Nervoso Centrale, Riv. sper. di Freniat. **36**:721, 1910.

7. Favorsky, A.: Die Abbauerscheinungen bei Tabes Dorsalis, Nissl-Alzheimer's Arbeiten, **6**:74, 1913.

NO DEGENERATION PROCESSES OBSERVED IN SUB-CORTICAL FIBERS

In spite of the marked ganglionic and interstitial changes, no degeneration of the subcortical fibers is found, nor are there any myelophages, gutter cells or other evidence of secondary glia changes. As in the cortex, the glia changes in the subcortical areas are of a progressive nature, represented by glia cells rich in protoplasm, or large-sized glia nuclei rich in chromatin. In the spinal cord there are, in addition, a great many astrocytes. The pons, medulla and spinal cord show the same intensity of interstitial changes as the large ganglia and midbrain. The spinal canal is usually occluded, either by proliferated ependyma



Fig. 12.—A destroyed ganglion cell from the subthalamic region surrounded by plasma cells. (Toluidin-blue stain, $\times 1,200$.)

cells, or by an amorphous mass mixed with lymphocytes and certain thin, elongated bodies stained dark with Bielschowsky's method (probably bacilli).

SUMMARY OF HISTOLOGIC STUDY

In summing up our histologic findings we may say that they concern the mesodermic (interstitial) and ectodermic (parenchymatous) elements. In the cortex, the parenchymatous changes predominate; in other regions (peduncles, internal capsule, lower portion of pons, bulb and cord) the interstitial changes predominate, while in the basal ganglia, substantia nigra and tegmental region of pons, both types are equally in evidence. In other words, the parenchymatous and inter-

stitial changes may exist independently of each other, and an absolute relationship between them cannot be claimed.

Both these large groups of pathologic changes are, according to Nissl and his school,⁸ essential in and characteristic of an encephalitis. It is needless to state that the above-described findings would satisfy

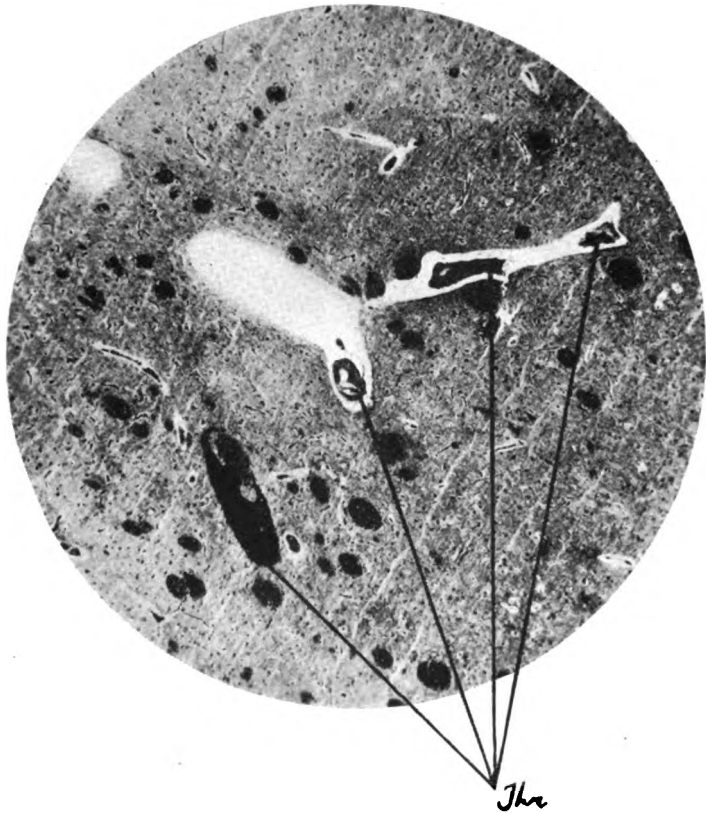


Fig. 13.—Nucleus candatus (its head bordering on the lateral ventricle). The numerous light channels inclosing vessels are so-called *Schrumppräume* of Nissl; the large black spots are bundles of white cortical fibers (*Haubenfasern*) of the cortex; the small dots are glia nuclei. Some vessels show so-called hyaline thrombi (*Thr*). (Bielschowsky stain, $\times 50$.)

even such a severe critic as Nissl that our three cases are instances of an encephalitis, resembling as they do in their pathologic features paralytic dementia, sleeping sickness and poliomyelitis.

By the absence of foci of softening, of large hemorrhages, of gutter cells, of polymorphonuclear elements, the lethargic form of encephali-

8. Ranke, Otto: *Beiträge zu der Lehre von Meningitis Tuberculosa*, Nissl-Alzheimer's Arbeiten 2: 1908.

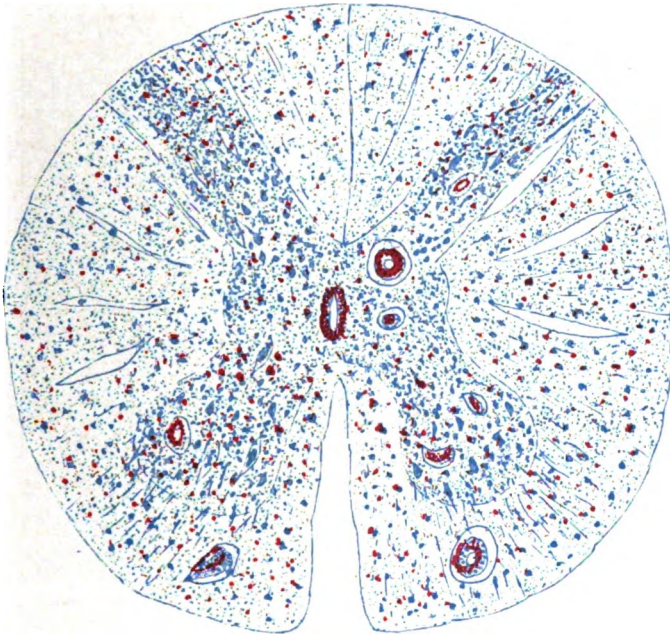


Fig. 14.—Spinal cord from Case 3 (lumbar region). The ependyma of the central canal, the blood vessels, the gray and white substances show an abundance of red spherules described in the text. (Frozen section; Alzheimer-Mann stain.)

tis, in our own cases at least, differs from other types, like Wernicke's polioencephalitis superior hemorrhagica,² acute hemorrhagic encephalitis,⁹ influenzal encephalitis,¹⁰ traumatic and experimental encephalitis (Friedman,¹¹ Coen,¹² Lottmar¹³ and many others). In Wernicke's

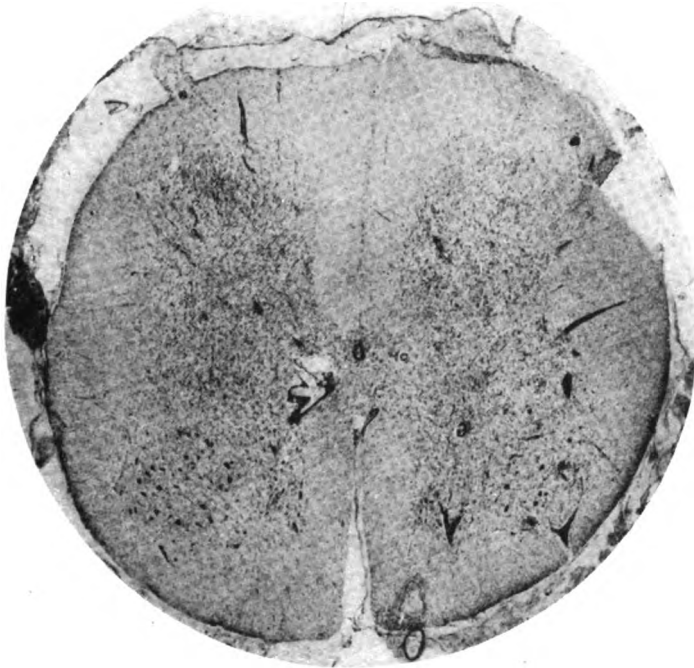


Fig. 15.—Lumbosacral portion of the spinal cord (Case 3). The membranes are slightly infiltrated; the subarachnoid space shows on the left a congested vein; the anterior and posterior horns exhibit a number of infiltrated vessels; the number of ganglion cells in the anterior horns is normal; the ependyma cells of the central canal are proliferated (a magnifying hand-glass will show the details better). (Toluidin-blue stain, $\times 16$.)

9. Deiters: Ueber Haemorrhagische Encephalitis, *Neurol. Centralbl.* **17**: 722, 1898. Friedman: *Deutsch. Ztschr. f. Nerven.* **16**:93, 1899. Brie: *Neurol. Centralbl.* **16**:2, 1897. Schmidt: *Deutsch. med. Wchnschr.* **18**:703, 1892. Sträussler: *Jahrb. f. Psych. u. Neurol.* **21**:253, 1902.

10. Bücklers: *Arch. f. Psych.* **24**:730, 1892. Königsdorf: Ein neuer Fall von acuter Haemorrhagischer Encephalitis waehrend der jetzigen Influenza-Epidemie, *Deutsch. med. Wchnschr.*, 1892, p. 182. Fürbringer: *Deutsch. med. Wchnschr.* **18**:182, 1892. Oppenheim, H.: *Deutsch. Ztschr. f. Nerven.* **6**:398. Oppenheim and Cassirer: *Encephalitis in Nothnagel's Encyclop.* **11**:2, 1907.

11. Friedman, M.: Studien zur Pathologischen Anatomie der acuten Encephalitis, *Arch. f. Psych.* **21**:461, 1890; also, *Neurol. Centralbl.* **8**:441, 1889.

12. Coen, E.: Ziegler's Beiträge zur Pathologischen Anatomie, etc. **2**:107, 1888.

13. Lottmar, Fritz: Beiträge zur Histologie der akuten Myelitis und Encephalitis, sowie verwandter Prozesse, *Nissl-Alzheimer's Arb.* **6**:248, 1913.

form and the so-called hemorrhagic encephalitis the hemorrhages are the principal feature with abundance of reactive phenomena on the part of the glia and total lack of parenchymatous, ganglionic changes. In fact, the characteristic signs of encephalitis, as outlined above, are absent in these two forms, which fact has been already pointed out by Biet,¹⁴ Redlich¹⁵ and Schroeder.¹⁶ These authors justly doubt whether Wernicke's polioencephalitis should be called encephalitis at all. The same can be claimed for the influenzal and other forms in which practically the only lesions are hemorrhages. In the traumatic and experimental forms, foci of softening or necrobiosis are common, which are, however, totally foreign to the lethargic type.

COMMENT

Our histologic studies thus have brought out the fact that the epidemic encephalitis distinctly differs from the condition previously described as influenzal encephalitis. Hence, if the influenzal encephalitis of previous authors really is influenzal, the form now under discussion decidedly is not. As stated before, it resembles the African sleeping sickness, dementia paralytica and the acute anterior poliomyelitis. The resemblance to the sleeping sickness is so great that a differential diagnosis on histologic basis is impossible. However, the sleeping sickness is chronic or subacute, while the lethargic encephalitis is rather acute in its course. The infiltrations of the pia and cortex are more pronounced in sleeping sickness. The slight difference in some pathologic changes (for instance, an occasional proliferation of vessels) may be explained by the longer duration of the African disease. It is also difficult to differentiate the brain changes of epidemic encephalitis from those of paralytic dementia. In the latter, however, the meningeal infiltration is very pronounced; the infiltration cells are mostly plasma cells, there is marked new formation of capillaries, a distinct proliferation of the intimal layer and of elastic fibers is present; all these changes being *especially pronounced in the cortex*.

As some of these characteristic features, like intima and elastica changes and proliferation of vessels, may be absent in acute cases of dementia paralytica and sometimes may be even more pronounced in the subcortical regions (the atypical forms of Lissauer and Alzheimer), it is clear that it may sometimes be difficult to differentiate these two diseases.

14. Biet: Contrib. a l'étude des affections nerveuses consecutives à la grippe, Paris, 1895. (Quoted from Friedmann, Footnote 9.)

15. Redlich, E.: Ueber Encephalitis Acuta, Centralbl. f. Allg. Path. 11:513, 1900.

16. Schröder, Paul: Zur Lehre von der Akuten Haemorrhagischen Polioencephalitis Superior (Wernicke), Nissl's Arb. 2:145, 1908.

In poliomyelitis the changes are largely confined to the spinal cord, the cerebral forms being very rare. Suffice it to say, that the spinal cord changes of Case 3, though much resembling those of acute poliomyelitis (vascular infiltration), are by no means identical with them. Hemorrhages, foci of softening, polymorphonuclear cells, marked satellitosis, so frequent and common in acute anterior poliomyelitis, are missing, while the parenchymatous and meningeal changes usually so marked in poliomyelitis are not striking in our case (Fig. 15).

The brief analysis of our findings shows a similarity in all essential points to those described in epidemic encephalitis by Marie,¹⁷ Marinesco¹⁸ and others.

CONCLUSIONS

1. The pathologic changes in epidemic (lethargic) encephalitis involve the entire central nervous system.

2. These changes are both parenchymatous and interstitial.

3. The former principally involve the cortex, the latter the peduncles, internal capsule, lower pons, bulb and cord. In the basal ganglia and midbrain both parenchymatous and interstitial changes are pronounced.

4. In its histopathology epidemic (lethargic) encephalitis differs from the various types of hemorrhagic encephalitis, but it is identical with that of African sleeping sickness (trypanosomiasis).

5. It differs from typical cases of paralytic dementia by the topography of the lesions and by absence of vascular wall changes, which probably are due to the more chronic course of the latter.

6. It also differs from the various types of traumatic and experimental encephalitis and from acute anterior poliomyelitis.

7. It is pathologically a distinct morbid entity, a disease *sui generis*, and does not resemble the conditions previously described as influenzal encephalitis.

8. The similarity between the pathologic changes in African sleeping sickness and epidemic encephalitis suggests a close relationship of their etiologic factors, that is, the epidemic encephalitis may be caused by a parasite akin to a trypanosome.

17. Marie, Pierre and Tretiakoff, C.: Examen histologique des centres nerveux dans deux cas d'encephalite lethargique, Bull. et mem. Soc. med. des hôp. de Par., May 24, 1918.

18. Marinesco, G.: Contribution a l'etude de l'histologie pathologique de l'encephalite léthargique, Bull. de l'Acad. de méd. 80:411, 1918.

DISCUSSION

DR. MAX H. WEINBURG, Pittsburgh: Speaking of the difference between what Dr. Bassoe found and the findings of the other workers such as Welsh and Marinesco regarding the differentiation of poliomyelitis and lethargic encephalitis, the findings are not in accord with those of the other workers. Marinesco pointed out that there are more plasma cells in the latter condition, and Welsh corroborated him. Marinesco pointed out that there is no neuronophagia or very little. This disease is of such wide manifestation that you will find a different pathology in the cases, just as clinically such cases manifest symptoms altogether different. There are many punctate hemorrhages found all over the brain. In one case we had in New York the lesion was believed to be a tumor. Most of the neurologists believed it to be a sarcoma of the brain. At necropsy a large hemorrhage can be explained readily by the fact that the disease in some cases is more marked than in others, and therefore there is some variance in results. Welsh reported several cases of the cord where there were cervical lesions, but in one case the lesion was as far down as the lumbar region. Clelland of Australia who has experimented on 120 animals points out mainly the perivascular infiltration and that the disease can be transmitted. The differential points between poliomyelitis and encephalitis lethargica are that there is no or little neuronophagia as there is in poliomyelitis, the plasma cells predominate, and there is little parenchymatous change, and it is for that reason that most of these patients recover. There is a prevailing belief that this is a disease of high mortality, but this is merely due to the fact that a great many cases are not recognized. I have had occasion to see over sixty cases in New York with a very low mortality.

DR. PETER BASSOE, Chicago: In regard to the question of cell changes, I can only relate my own experience. At the time I reported my first two cases I stated that hemorrhage was marked and there were no cell changes. After making a thousand checks we found the hemorrhage was an accidental finding, I happened to strike the only hemorrhage the first time. You must make sections all through and conduct your investigation with an open mind. I find hemorrhages are not a feature and neuronophagia is. It is, to be sure, not overly profound. You can look at a dozen sections, particularly of the cortex, without finding it, but deeper down you will not miss it. I have now a brain from a patient who had an acute encephalitis, and there is an extensive condition throughout the brain. It may be a true influenzal encephalitis. I am not prepared to say anything about the relation of this disease and influenza; only if what has previously been described is influenzal encephalitis, then this is not. As to the Australian cases, we have to be a little slow, as we do not know what they were. Many of the reports look like poliomyelitis, and one of the foremost investigators there believes the cases were poliomyelitis.

GENERAL PARALYSIS TREATED BY INTRAVENTRICULAR INJECTION OF ARSPHENAMIN

REPORT OF THE NECROPSY FINDINGS IN A CASE *

IRVING J. SANDS, M.D. (BROOKLYN)

First Lieutenant, M. C., U. S. Army; Assistant Physician in Charge of the Pathological Department of the Manhattan State Hospital, Ward's Island, N. Y.; Instructor in Neurology, College of Physicians and Surgeons

Following the discovery of the *Treponema pallidum* in the brain in cases of general paralysis, and encouraged by the reported favorable results obtained in the treatment of neurosyphilis by the intraspinal injection of arsphenamin as advocated by Swift and Ellis and by Ogilvie, many men began to treat general paralysis with arsphenamin, administering the drug either by the intravenous or intraspinal method. The results obtained are now well known to those who have followed the evolution of therapy of neurosyphilis since the introduction of arsphenamin. Failing to obtain as favorable results from the use of arsphenamin in paresis as from its employment in the more benign forms of neurosyphilis, and stimulated by the results from animal experimentation, Hammond and Sharpe¹ introduced a method of injecting arsphenamin into the ventricles of the brains of the cases of general paralysis, hoping to get the drug more closely to the site of the pathologic process. They reported a series of cases which showed clinical improvement following this mode of treatment. Many observers were impressed with these results, and Neymann and Brush,² in their analysis of the different forms of treatment of paresis, spoke very favorably of this method.

The following case is of interest as it is the first case which has received this form of therapy, and has been studied clinically as well as at the postmortem examination.

* From the Department of Pathology, Manhattan State Hospital, Ward's Island, N. Y.

* Presented before the New York Neurological Society, Oct. 1, 1918.

1. Hammond, G. M., and Sharpe, Norman: The Treatment of Paresis by Injections of Neosalvarsan into the Lateral Ventricle, J. A. M. A. **65**:2147 (Dec. 18) 1915.

2. Neymann, C. A., and Brush, N. H.: The Treatment of General Paresis, Arch. Int. Med. **22**:245 (August) 1918.

REPORT OF CASE

History.—H. F., a man aged 40, was born in New York City of German parentage. He was graduated from the public school. He married in 1905 and has one living child; no miscarriages. He drank early in life, but he was a good provider for the family. He was of a happy disposition and was well liked by his friends. He has had several attacks of rheumatism. No knowledge of any venereal diseases. Worked satisfactorily as a carpenter. In December, 1915, he showed the first evidence of the disease, becoming forgetful. He continued to work until the middle of 1916, when his memory became so poor that he had to discontinue his work. At the same time he became irritable, showed speech defect, and began to manifest unsteadiness in walking. He later became grandiose in his statements, saying that he was a millionaire, etc. He finally became very restless and was sent to the Post-Graduate Hospital for treatment.

Clinical Course.—In March, 1917, he received one intraventricular injection of arsphenamin, through a trephined opening in the skull. Subsequently he was transferred to Bellevue Hospital, where he was characterized by the physicians as being simple, childish, dull in speech; wandered about in an aimless manner; was very irritable; was expansive in his statements; his pupils were small and irregular and did not react to light; knee jerks were increased.

Examination.—He was admitted to the Manhattan State Hospital on May 16, 1917. An examination in this institution revealed considerable mental deterioration; he said he was a carpenter and that he was feeling fine, and that he had to go to work. He did not know when he was married. He was disoriented. He had no memory on immediate past. He did small calculations very poorly. His speech showed considerable defect. He could not interpret what he had read. He had no insight. He expressed grandiose ideas. His judgment was poor. Physically, he showed good nutrition. Pupils were irregular, unequal and reacted very sluggishly to light. Spinal fluid showed positive globulin, 54 cells, and a ++++ Wassermann reaction.

Subsequent History.—He soon began to soil himself and became quite ataxic. He was very euphoric in the latter part of May, and his speech became so defective as to be hardly understood. On June 27 he developed convulsions and died on the following day.

ANATOMIC SUMMARY

Necropsy performed fifteen hours postmortem. The remains of a well developed, and of a moderately well preserved man appearing about 40 years of age. Rigor mortis was present in all extremities. Excepting slight abrasions over the sacrum and over the right knee, there was nothing of significance noticed in the skin of the body. Over the junction of the parietal and frontal bones, just to the left of the midline, a distinct depression of the scalp was seen. This was due to a defect in the subjacent bone evidently caused by a trephined area in the skull, 3 by 2 cm. in size. The scalp at this area was adherent to the subjacent dura. The calvarium stripped with considerable ease, and left a pale dura beneath it. Immediately beneath the trephined area in the skull there was a semicircular incision in the dura; the

resultant flap in the dura was half the size of a 5-cent piece. The edge of the flap was adherent to the subjacent pia-arachnoid by only very thin fibrous tissue, and it could readily be lifted from the underlying tissue. The dura as a whole appeared quite pale, and over the anterior



Fig. 1.—Note thickened pia and characteristic appearance of exudate. Left hemisphere more involved than right. The defect in the right frontal lobe was caused by removal of a section for fixation in alcohol. The pia-arachnoid flap is in the plane of the arrow.

third of the brain, most marked over the frontal poles, it was decidedly puckered and redundant. There was no pathologic condition found in the dura. The vessels in the dura were of the normal size and

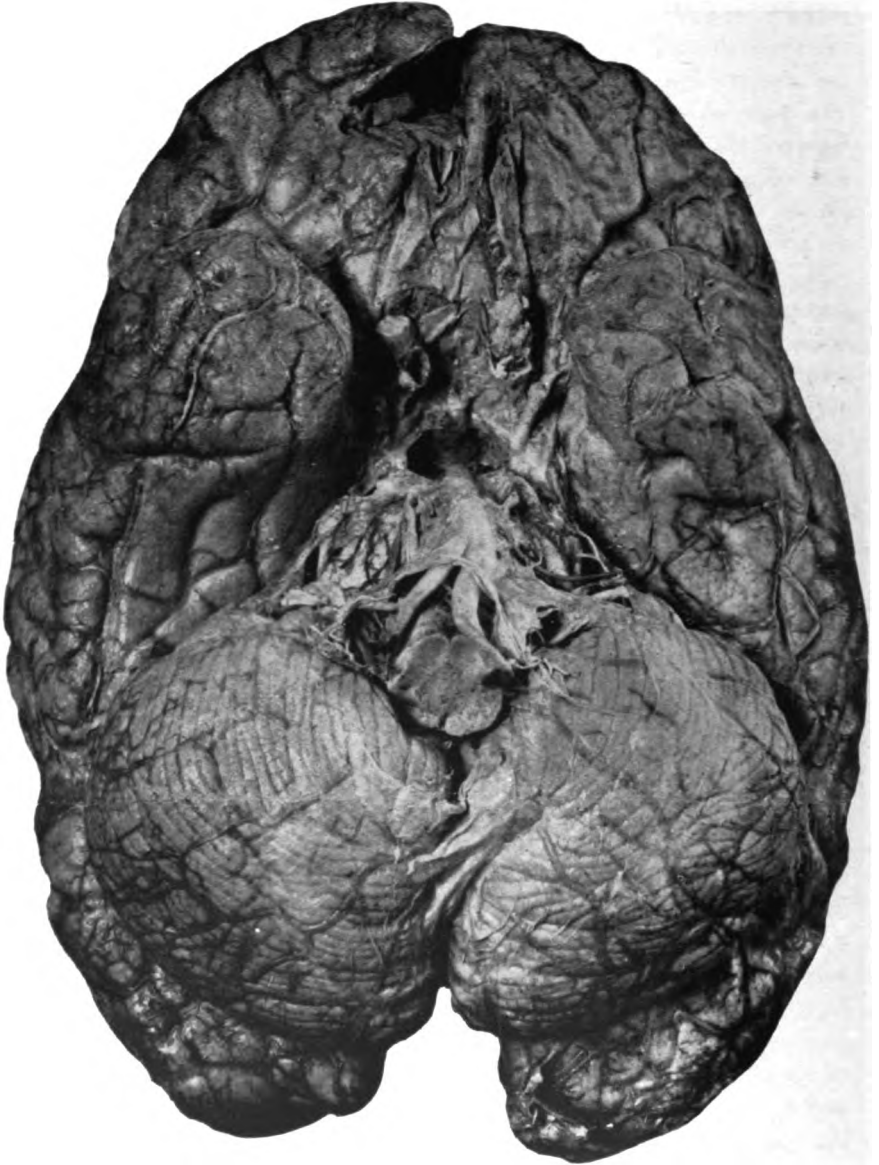


Fig. 2.—Note exudate on the base of the brain. Lesion is greater on the left side. Note exudate over the left side of the cerebellum. The defect in the right gyrus rectus was caused by removal of section for fixation in alcohol.

appearance. On incision of the dura and arachnoid an excessive amount of cerebrospinal fluid escaped.

The brain weighed 1,175 gm. It was of medium size, and its consistency was about normal. The pia over the anterior two-thirds of the brain showed a characteristic milky cloudiness and thickness; over the posterior third of the brain the pia was fairly clear and glistening. The base of the brain, especially over the interpeduncular spaces, showed thickened and clouded pia. The pia over the left hemisphere was decidedly more thickened and evidently more involved than the pia over the corresponding parts of the right hemisphere. The pia over the cerebellum, and also in the ponto-cerebellar angles, was of a milky, clouded appearance. On lifting the flap of dura immediately beneath the trephined area in the skull there appeared a part of the brain cortex, evidently due to the adhesion of the dural flap to the subjacent pia-arachnoid. Characteristic granulations were seen in the gyri recti and in the ventricles, especially on the floor of the fourth ventricle. There was considerable atrophy in the frontal lobes, the convolutions being well rounded out and the sulci and fissures were quite wide and shallow. The large vessels, namely, the basilar, internal carotids, and the cerebrals were quite normal in their macroscopic appearance. No hemorrhagic areas were noticed.

Sections from the superior frontal, precentral, paracentral and occipital convolutions, from the gyrus rectus, and from the lobule immediately beneath the trephined area, and from the medulla and cerebellum were examined microscopically. The pia showed considerable edema; there was present the characteristic lymphoid and plasma cell infiltration, especially about the pial vessels; numerous mast cells were also seen; the exudate was greater in the sections from the left hemisphere. The neuroglia layer was thicker than normal, and throughout the cortex there was noted an increase in neuroglia cells; in several places there were typical pictures of satellitosis. The cortex showed considerable disorganization; there was much edema; the cells were found in all stages of degeneration and characteristic pictures of cloudy swelling and granular degeneration were noticed, especially in the sections from the left hemisphere. The perivascular exudation of lymphoid and plasma cells was quite striking. Numerous mast cells and a small number of rod cells were also noted. The sections from the cerebellum and from the occipital lobule showed very slight pathologic changes as compared with the sections from the other regions studied. The sections from the left hemisphere showed more edema, greater exudation and a more advanced cortical disorganization than the sections from the right hemisphere. The spinal cord showed nothing of significance macroscopically; but microscopically,

considerable degeneration was noticed in the fibers of the posterior columns stained by the Marchi method. Sections from the right optic nerve showed many lymphoid and a few mast cells running through the nerve. The other cranial nerves showed no significant changes macroscopically. The internal ears were opened and found in normal condition.



Fig. 3.—Note infiltration of pia with lymphoid and plasma cells; considerable edema of pia is present. Note increase in neuroglia cells in the adjacent cortex.

The *pituitary* gland did not present anything of significance. The *thyroid* gland was of normal appearance; the alveoli were distended with colloid and the cells were definitely flattened. The *thorax* was externally symmetrical; the sternum stripped with ease; the costal cartilages were not calcified. Both pleural cavities were free from fluid, but each contained moderately dense adhesions in the apices.

The *right lung* weighed 580 gm. and the left 560 gm.; the apices showed definitely healed tuberculous nodules which had been partly replaced by calcium salts, and between these nodules there were numerous strands of connective tissue; the posterior portions of the lower lobes showed distinct edema and bronchopneumonic patches.

The *pericardium* was free from fluid and adhesions. The *heart* weighed 330 gm.; it was covered on its anterior surface by a very

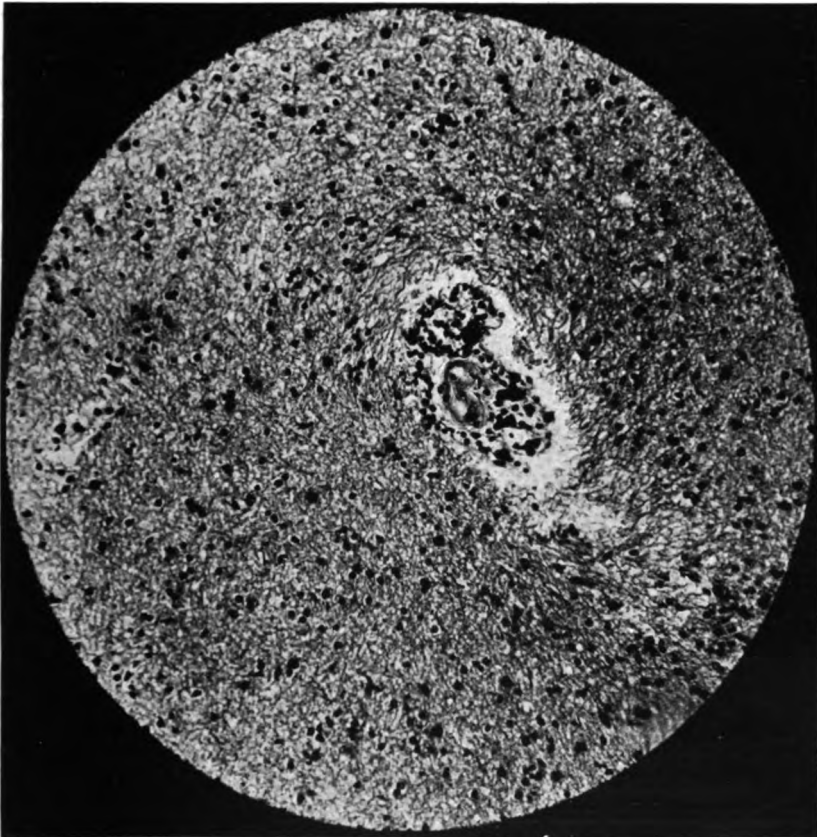


Fig. 4.—Note the perivascular exudation of lymphoid and plasma cells around two small cortical vessels. There is also seen an increase in neuroglia cells in the cortex.

thick fat pad; it was of normal size and consistency; all valves were compensating and their cusps were quite delicate; the papillary muscles were normal in appearance; the coronaries were patent. The *aorta* presented a characteristic picture; the arch was definitely dilated; there was a decided loss of elasticity in the entire aorta; throughout the thoracic portion there were numerous linear scars and pearly nodular

elevations; the abdominal portion was relatively free and only an occasional yellow-colored ulcerated area was seen; microscopically, the intima presented a fairly normal appearance; the media was infiltrated with many lymphoid and plasma cells which in places were grouped and assumed a picture which was highly suggestive of miliary gummas, the adventitial vessels showed perivascular exudations of lymphoid and plasma cells.

The *abdomen* was scaphoid; the panniculus adiposis was of medium size and of normal color; the peritoneal cavity was free from fluid; the omentum extended into the pelvis and in the left hypochondrium it was adherent to the peritoneum by thin but firm adhesions. The appendix was entangled in a mass of adhesions; it had an injected inflammatory appearance. The *stomach* was distended with gas and was otherwise of a normal appearance; the rest of the intestinal tract showed nothing of importance. The *spleen* weighed 130 gm.; it was of a normal size and appearance, and of a slightly increased consistency. The *liver* weighed 1,450 gm.; it was somewhat larger than normal, and its color was definitely yellow and its consistency was diminished; microscopically, the cells showed a condition of fatty infiltration and cloudy swelling. The gallbladder was distended with bile; all ducts were patent. The *pancreas* showed nothing remarkable. The *adrenals* were of normal size and consistency, and on section presented a normal cell layer arrangement.

The right *kidney* weighed 110 gm. and the left 140 gm.; the right appeared normal, but the left kidney showed a few adhesions between the capsule and the cortex, and the cut section presented a rather boiled appearance. The ureters showed nothing remarkable; the bladder was contracted. The genitals appeared normal.

ANATOMIC DIAGNOSIS

Trephined opening in the skull, diffuse meningo-encephalitis, bilateral bronchopneumonia and pulmonary edema, syphilitic aortitis, chronic appendicitis and fatty liver.

SUMMARY

A man, aged 40, married, who had one living child, a negative family history, but an alcoholic personal history, who had no knowledge of any venereal disease, began to show lapses of memory in December, 1915, which increased in severity so that he was obliged to discontinue his work as a carpenter in the middle of 1916; he then became irritable, showed speech defect and became ataxic; in March, 1917, he received one intraventricular injection of arsphenamin through a trephined opening in the skull. He was admitted to the Manhattan State Hos-

pital in May, 1917, when he showed disorientation, poor memory, expressed grandiose ideas; showed marked speech defect, pupils were unequal and reacted very sluggishly to light, and the spinal fluid showed positive globulin, 54 cells, and a positive Wassermann reaction. He soon began to soil himself, became very euphoric and died following a convulsion. The postmortem examination showed the characteristic lesions of general paralysis in the brain; namely, a thickened pia showing a characteristic milky exudate in the anterior two-thirds, atrophy of the anterior poles of the cortex, lymphoid and plasma cells infiltration into the pia, neuroglia increases, cortical disorganization and perivascular exudation of lymphoid and plasma cells, many mast cells and a few rod cells, granulations on the floor of the ventricles, syphilitic aortitis and bilateral bronchopneumonia and pulmonary edema; the lesions in the left side of the brain which received the arsphenamin were more intense than those in the right side.

312 Hopkinson Avenue.

I am indebted to Dr. M. B. Heyman for allowing me to present and publish this case, and to Dr. F. C. Wood for the photomicrographs.

CONGENITAL TUMOR OF THE BRAIN (TELANGIECTASIS) AND ASSOCIATED CEREBRAL MOVEMENTS *

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Professor of Neurology, University of Pennsylvania

PHILADELPHIA

The occurrence of congenital telangiectasis of the brain, diagnosed clinically and treated by operation is infrequent, and for this reason I have made a study of the case here recorded. It affords the opportunity to discuss associated movements of the limbs resulting from cortical defect.

REPORT OF CASE

History.—J. C., aged 12, was admitted to the University Hospital, June 17, 1918. The mother of the boy was exposed to roentgen rays when three and one-half months pregnant with this boy. Another child was held on the lap of the mother while it was being roentgen rayed. The birth of the patient was normal. When he was 2 or 3 months old his right upper limb was observed to be paralyzed, but it probably had been so from birth. The mother noticed when the child was about 1 year old that the right lower limb was paralyzed, because the child made no effort to crawl or stand. Since his birth he has had a telangiectasis of his forehead over the left eye, one in the left side of the scalp near the front hairline, a similar one in the left eyeball, and a small one on his back.

The first convulsion occurred May 1, 1918. This implicated the whole body and was with unconsciousness. The second attack occurred four weeks after this one and was general, and similar convulsions occurred later. He learned slowly at school and was backward in his classes.

Examination.—He wrinkles his forehead slightly. He closes both eyes well together, but cannot close either separately. In showing his teeth some weakness of the right side of the face is detected. The tongue is protruded awkwardly. He cannot close the right hand alone at all, but when he closes the left hand there is associated movement of the same kind with the right hand which the boy cannot prevent. Flexion at the left wrist and often even at the elbow produces a similar movement of the corresponding part of the right upper limb. The voluntary movement of the right upper limb at the elbow and shoulder is impaired. The movement at the right wrist is lost except in association with movement at the left wrist. The right hand is flaccid. The dynamometer registers 2 degrees in the right hand, 10 degrees in the left hand. The right upper limb is poorly developed and the right hand is much smaller than the left. Diadokokinesis is normal in the left hand and the test does not produce associated movements in the right hand. Stereognostic perception is lost in the right hand and the boy can tell only whether the object is cold or warm with this hand. It is normal in the left hand.

* Read before the Section on Nervous and Mental Diseases at the Seventieth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1919.

He has imperfect movement in extension of the right lower limb and the power is not great. He cannot dorsally flex the right foot. Associated movements are present in the right toes. He cannot move the right toes alone, but when he moves the left toes the right toes move similarly, but associated movements are more pronounced in the right hand than in the right foot. He walks without difficulty.

Achilles and patellar tendon reflexes are exaggerated on the right side, normal on the left side. The Babinski reflex is doubtfully present on the right side, absent on the left. Temperature, touch and pain sensations are normal.

Operation.—Dr. Frazier operated on the patient June 26, 1918. A flap was made over the Rolandic fissure. On reflecting this, a dark-bluish discoloration with bulging of the brain appeared in the anterior and superior third of the opening, as though cavernous spaces of blood were beneath the dura at these places. The branches of the meningeal vessels were three or four times as large as they usually are and bled freely from the time the osteoplastic flap was reflected, and closure was attended with much difficulty. On incising the dura along the superior margin of the opening a tremendous venous channel was penetrated in which the blood welled until hemostatic clamps were applied. So much blood had been lost from various causes that no attempt was made to explore further.

Results.—July 24, 1918: The father reported that no convulsion had occurred since the operation. The boy has no increase of the right-sided weakness. He had a number of convulsions on July 29, but only one attack from this date to September 9.

April 11, 1919: His mother stated that his last convulsion had occurred in October, 1918, and was very slight. He had had no attack of any kind since and had been perfectly well, and had done well in school. Whereas formerly he had obtained very poor marks and had been observed to be deteriorating mentally, now he was bright and had recently obtained an almost perfect grade.

CASES MENTIONED IN THE LITERATURE

Angiomas have been classed by some as congenital tumors. The first case reported by Oppenheim in his paper on congenital brain tumor is the most interesting of his series. The patient had left hemianopsia since birth or early childhood. Sight had been absent in the left fields as long as he and his mother could remember; as a child he had not seen food or toys placed on his left side, and yet he seems to have had no other signs of cerebral disease until he was 18 years old. At that age he had severe headache, paresthesia, paralysis of the entire left side, etc. These symptoms disappeared leaving some weakness of the left side. When 25 years old he had a second attack consisting of severe headache and vomiting and lasting about 14 days. At 26 years he had a third attack similar to the first and became paralyzed on the left side. The diagnosis of a lesion of the right optic thalamus and the adjoining region was made. It was supposed to be a congenital tumor which caused focal symptoms at different periods because of transitory swelling or hemorrhage, probably an angioma cavernosum.

In three out of Oppenheim's four cases the symptoms began in adult life, in one at 18 years, in another at about 23 or 24 years, and in the third at about 33 years. The relative late onset of symptoms in angioma is striking, also the possibility of repeated hemorrhage in such a tumor, but all of his cases seem to have been without either operation or necropsy. An angioma of the face was found in two of the four cases, and Oppenheim mentions that experience has shown such a lesion is frequently associated with angioma of the cerebral meninges, and he refers to Kalischer, Emanuel, Bruns and himself in this connection.

He mentions as congenital tumors of the brain dermoid cysts, teratoma of the hypophysis, angioma, possibly cholesteatoma and neuroma of the brain nerves.

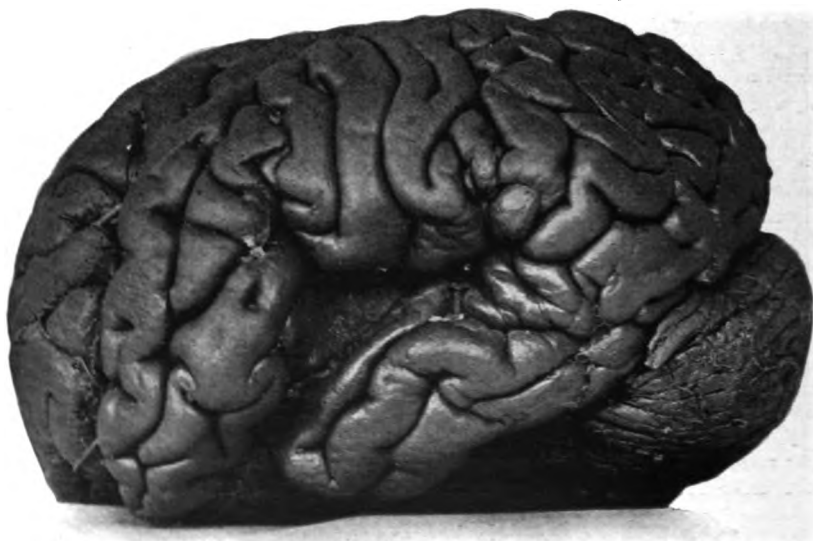


Fig. 1.—Porencephaly of the left cerebral hemisphere.

These congenital tumors may cause symptoms in early childhood, and then be latent many years, or produce symptoms with long intermissions, or the symptoms may first appear in later life.

Oppenheim had only twice been able by biopsy to confirm the clinical diagnosis of angioma of the brain, although he had observed angioma a few times at operation in cases in which he had made the general diagnosis of brain tumor. He does not distinguish sharply between angioma and telangiectasis.

In the first of the two cases reported by Ernest Sachs there were fever and convulsions confined to the left side of the body beginning three months after a fall. The possibility of hemorrhage from the

telangiectasis is suggested by this, although Sachs does not accept this as a factor. A left-sided paralysis developed also and disappeared in a month. Then the patient was well about one year and because of the reappearance of jacksonian convulsions an operation on the brain was performed. A huge bluish mass of vessels protruded when the dura was opened, and was so dense in places that the underlying cortex could not be seen. An attempt to remove this mass at a second operation resulted fatally.

The second case was that of a patient with a pale telangiectasis over the forehead. An angiomatous process was found in the dura, which on reflecting the dura was found to have numerous connections with the pial vessels. All the vessels connecting with the pial vessels were ligated and the dural mass was ligated above and below. Two days after the operation the patient had a violent convulsion, but since then had been well. It would seem as though this extensive ligation of the vessels had not had any permanent serious result.

Sachs found very few cases in the literature like his second case. He mentions that the process in all three of Cushing's cases was in the dura, and that Cushing does not mention the possibility that an identical process might occur on the cortex.

Sachs disputes the propriety of calling these tumors angiomas and quotes Virchow and Adami in support of this opinion. He emphasizes that a telangiectasis is a congenital dilatation of capillaries without any new-formed blood vessels, while an angioma is a new growth and only properly so-called when new blood vessels are formed. These distinctions are lost sight of in the literature. He also refers to the existence of telangiectasis elsewhere on the body, especially the face, and mentions that this was true of Pean's case and of Cushing's.

In the case reported by Castex and Bolo the lesion was designated by them as *angioma venosum racemosum* and extensive ligation of the vessels was followed by recovery. No writer so far as I know has spoken of the danger of extensive ligation, and yet I have seen serious symptoms result in two cases from this procedure. In one case a hemorrhage occurred into the brain, and in the other hemiplegia resulted. I would therefore suggest caution in tying many vessels at one time, in cortical telangiectasis.

In my patient the numerous angiomas of the surface in connection with symptoms of cortical lesion indicated that the latter probably also was of the same vascular character, and I advised operation with this probability in mind. The operation revealed extensive telangiectasis of the motor cortex. Caution was exercised by Dr. Frazier to avoid ligating too many vessels, and no complications occurred. I have observed two cases of teratoma of the region of the medulla oblongata both with successful operation — one by Frazier and one by Elsberg.

The associated movements of the right side, the paralyzed side, were very marked in the case I have described. Such movements are well known and are most pronounced when the paralysis has dated from birth. Very rarely associated movements occur in people otherwise normal, who are unable to use either hand without a corresponding movement of the other hand, and cases of this character are attributed to Damsch, Fragstein, and Brissaud and Sicard by Lewan-

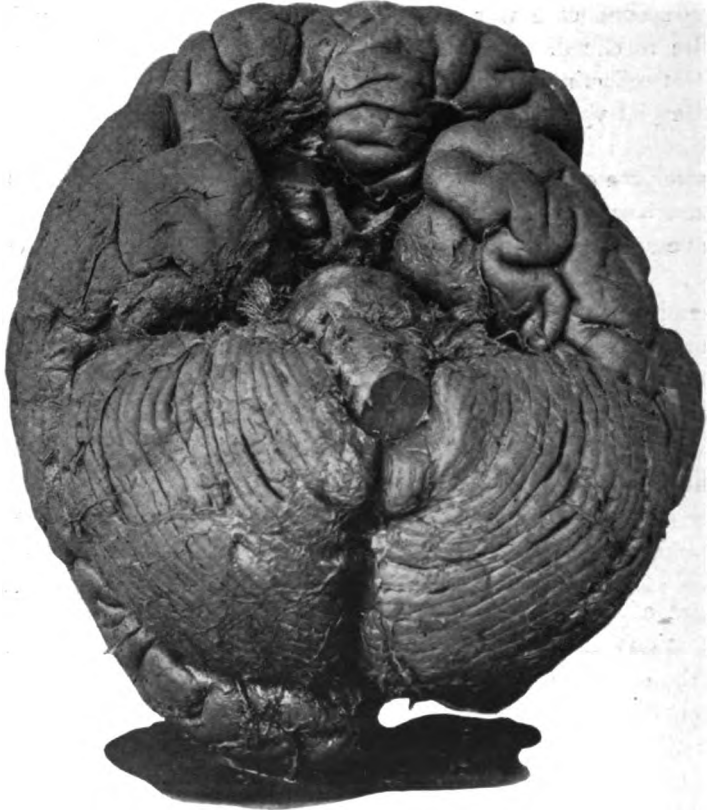


Fig. 2.—Another view of porencephaly of the left cerebral hemisphere.

dowsky. I have seen two striking examples of this: one case was presented by Drs. C. W. Burr and C. B. Crow before the Philadelphia Neurological Society; the other was exhibited at the last International Medical Congress held in London, but I have never seen any report of this case published.

The case presented by Burr and Crow was as follows: The patient, a man, had had the condition since infancy. Whenever he made any complicated voluntary movements with the fingers or wrist of either

hand the other hand involuntarily made the same movements. Very simple motions such as flexing or extending one finger could be made independently, but even then, as a rule, there was a similar simultaneous movement of the corresponding finger of the other hand. The condition was confined to the hand muscles and did not appear when the upper arm alone was used. The feet and toes were not affected. By strict attention he could prevent the unused hand from moving, but he could not, for any length of time, keep his attention fixed on what he was doing and on inhibiting the hand. He did his work as a seaman well until he was ordered to climb a rope. He demurred, and it was found that on letting go with the lower hand to reach for a higher grip on the rope he let go with the upper hand and fell.

In discussing this case, I suggested that each hand of this man was innervated from both sides of the brain equally or both were innervated from one side of the brain alone.

AUTHOR'S OBSERVATIONS

The central nervous systems from two cases in my possession throw light on the mechanism of these movements. Unfortunately, the clinical history is very deficient in each case, and I do not know whether associated movements were present or not. Both established the fact that the anterior pyramid on the side opposite to the lesion may be unusually large when the other anterior pyramid is absent from a congenital defect of the pyramidal tract of one side.

One was a case of *porencephaly*. The brain was from a low-grade boy, whose memory was fairly good, who sang, but never learned to read. He was partly paralyzed on the right side and never used his right hand. An opening extended into the left lateral ventricle, and the walls of this ventricle were continuous with the external surface of the brain (Fig. 1), and the base of the left frontal lobe presented a condition of agenesis. The anterior pyramid on the left side was absent (Fig. 3) so that no motor fibers could have passed into the cord from the left cerebral hemisphere through the anterior pyramid. The right anterior pyramid was unusually large. The fillet on the left side also was much smaller than that on the right side. Inasmuch as he was reported to have been only partially paralyzed on his right side, whatever limited voluntary power he had on this side must have come from the right cerebral hemisphere.

The second case was that of a man (G. S.) aged 66, who entered my service at the Philadelphia General Hospital, Jan. 24, 1916, a few days before I went off duty, and was never studied clinically by me.

He had left-sided palsy said to have dated from birth, but this had not prevented him from working. He was able to go about, but had very little use of the upper limb, and could not use the left hand.

Shortly before Christmas, 1915, he seems to have had a stroke, and he lost power in both lower limbs. His mentality was much impaired. The grip in the right hand was good.

A note was made Feb. 25, 1916, that he had partial paralysis of his upper limb and of both lower limbs, and was slowly regaining strength in the lower limbs. He died Aug. 2, 1916. A small area of softening was found in the upper part of the left lenticular nucleus and above it and was probably the cause of the attack in 1915. The lateral ventricles were dilated, but the right one was very much more so than the left. The right anterior pyramid was exceedingly small, while the left was abnormally large. The right lemniscus was also much smaller than the left.



Fig. 3.—The anterior part of the medulla oblongata from the case of porencephaly. The left anterior pyramid is wanting and the right anterior pyramid is unusually large. The left fillet is much smaller than the right.

The cervical region, on the whole, showed diminution in size of the left anterior horn and of the number of cells it contained. There were sections in which this distinction was not so evident. The left lateral column was considerably smaller than the right in the cervical region, but showed no signs of overgrowth of neuroglia. In the lumbar region it was impossible to make any such distinctions between the two sides of the cord. There was, therefore, a moderate hypoplasia of the left side of the cord in the cervical region.

In both these cases there was partial hemiplegia dating from birth, and in both the anterior pyramid on the side opposite to the lesion was

unusually large, while the other anterior pyramid was entirely absent in the case of porencephaly and exceedingly small in the case of hydrocephalus. It is reasonable to conclude that the intact pyramid contained homolateral motor fibers, and in this way some movement was still possible in the hemiplegic side. In 1895, while working in Dejerine's laboratory I made a study of the spinal cords in many cases of hemiplegia in which death followed the lesion in a comparatively short time—that is, in a period permitting the use of the Marchi method of staining—and was able to demonstrate degenerated fibers in both crossed pyramidal tracts, more in the side opposite to the lesion, in many of the cases, from an unilateral lesion of the cerebrum. This was evidence that the degenerated fibers came from the side of the brain in which was the lesion and indicated that each side of the brain supplied both crossed pyramidal tracts of the spinal cord, though in unequal degree.

COMMENT

It is interesting to note that the boy with telangiectasis of the cortex and pronounced associated movements developed convulsions which were not jacksonian but implicated both sides of the body. Probably these convulsions, like the voluntary and associated movements, originated in the right motor cortex. The loss of stereognostic perception of the right hand might be attributed to the lesion of the left parietal lobe, and it is interesting to recall that the lemniscus on the side of the cerebral lesion in the two cases with necropsy was very much smaller, as though the decrease in size might have been a form of retrograde degeneration or possibly a hypoplasia. I have seen a diminution in size of the lemniscus in several cases of lesion of the cerebrum.

The diminution of the whole right side of the body and limbs of this boy probably was in association with defect of the motor tract from the left cerebral hemisphere. The case of hydrocephalus showed some diminution in the size of the motor tract in the spinal cord from the right side of the brain. It is common to find arrest of muscular development in the paralyzed side in early acquired or congenital hemiplegia, and probably this is dependent on defect of the motor supply of the corresponding side. At birth the muscles may be developed independently of the motor nervous system, as von Leonowa first showed, but at a very early period of extra-uterine life—exactly at what period is not known—the motor nervous system takes control of the muscular system, and defect of the former leads to defect of the latter.

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DISCUSSION

DR. H. H. HOPPE, Cincinnati: In a large experience I have seen but one case of telangiectasis of the cortex. This occurred in a boy previously well up to the age of 19, when he developed attacks of jacksonian epilepsy. The most careful examination carried on over a period of from three to four weeks failed to reveal any signs of disease of the brain as manifested through physical signs. There was no weakness of the right side of the body, and not even the slightest indication of the Babinski sign of the sole of the right foot. On account of the fact that the boy had jacksonian seizures, it was deemed advisable to lay open the cortex and this condition of telangiectasis was found. In the majority of cases that I have read of there is no evidence to show before operation what is going to be found in the cortex. In the second place, I wish to question seriously after you have found this condition of telangiectasis whether it is wise to do anything further or not. The danger of ligating the vessels which supply the cortex of the brain has to be considered. In the case I referred to, the boy was made distinctly worse by the operation. He developed a hemiplegia which prevented him from earning his living, which he had been able to do before, and the operation did not improve his epileptic seizures. In fact, six months afterward they returned, and were just as bad if not worse than they had been before. While we may be justified in laying open the cortex to establish the diagnosis under these conditions, I doubt whether it is wise to tie off the arteries.

DR. WILLIAM G. SPILLER, Philadelphia: I think it is better to tie only a few vessels at the time of operation on telangiectasis of the brain, and, if necessary, to tie other vessels at a later operation. My patient was undoubtedly improved by operation, although recurrence of symptoms is possible. Often angioma of the brain cannot be diagnosed clinically, but angiomatous growths on the head or face should suggest that the lesion in the brain may also be angioma.

TREATMENT OF NEUROSYPHILIS BY ARSPHENAMIN INTRAVENOUSLY AND AUTO-ARSPHENAMINIZED SERUM INTRASPINALY*

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For the past seven years cases of syphilis of the central nervous system admitted to Dr. S. W. Lambert's service at St. Luke's Hospital have been treated with arsphenamin intravenously and the intraspinal administration of auto-arsphenaminized serum. About fifty such cases have received this treatment during this period, the majority of which have been of the tabetic type. The twenty-one cases reported herein have been investigated and followed up to determine the efficacy of this type of treatment.

TECHNIC OF ADMINISTRATION

The technic employed is essentially that of Swift and Ellis as described in their original article on the subject. Neoarsphenamin has been the drug usually administered intravenously; the other products, such as neodiarsenol, arsenobenzol, and novarsenobenzol, have been used when the former drug became difficult to obtain. Nine decigrams of neoarsphenamin were administered intravenously. One hour later sufficient blood was withdrawn to obtain from 10 to 30 c.c. of serum. The serum thus obtained was centrifuged for one-half hour and inactivated at 56 C. for one hour and injected intraspinaly the following day. In the early cases this serum was diluted with saline solution to 40 per cent., but since then the concentrated serum has been used.

REACTIONS OFTEN FOLLOW

Reaction to the intraspinal treatment has varied a great deal. The usual reaction has consisted of darting pains in the legs, beginning an hour or two after the introduction of the serum, and lasting from twelve to seventy-two hours. Associated with these pains there has sometimes been severe headache, girdle sensations, and disturbances of bladder control. The reaction in one patient (Case 3 in the table) was unusually severe. This patient, several hours after the intraspinal treatment, suffered from intense headache and severe lightning pains and loss of bladder control. His temperature rose to 101.2 F.,

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and he developed rigidity and retraction of the neck and a positive Kernig's sign. The following morning his white blood count rose to 31,000, with 95 per cent. of polymorphonuclear cells in the smear, and the cell count in the spinal fluid was 10,000, with 85 per cent. of polymorphonuclears. Cultures and smears of the spinal fluid were negative bacteriologically. Symptoms continued severe for five days and gradually abated. In a week the spinal fluid cell count fell to 75, and a month later it was 10, with 100 per cent. lymphocytes in the smear, and the patient was free from all pain or other after effects.

While in some instances very little discomfort is experienced, it is usual to have more or less discomfort for twelve or more hours following the intraspinal treatment. For this reason the following medication is ordered as a routine measure after such treatments:

	gm. or c.c.	
R Codeinae sulphatis.....	033	gr. ss
Acetanilidi	132	gr. ii
Acidi acetyl-salicylici		
sodii bicarbonatis..... āā	66	gr. x
M. et fac pulveres v.		
Sig.: A powder every three hours, for three doses, if needed.		

We have been unable to demonstrate any relationship between the severity of the reactions and the dilution, or quantity of the serum injected, in amounts between 10 and 25 c.c. We are convinced, however, that cloudy serums due to the presence of an excess of fat or to hemoglobin are much more likely to give severe reactions than are clear serums. The presence of hemoglobin can be guarded against in part by the use of dry syringes, needles, and flasks, and care in seeing that the blood is not shaken up in transit to the laboratory. The presence of an excess of fat is more difficult to control, but the serums may be improved in this respect by having the patients fast for five or more hours before the blood is to be withdrawn.

SYMPTOMS IN THIS SERIES OF CASES

In the accompanying table the data concerning the symptoms presented in these cases have been recorded under three main heads: subjective and objective symptoms, and laboratory evidence. Subjectively, the most common symptom has been pain; after this, disturbances in gait; then disturbances in bladder control and vertigo. Objectively, 85 per cent. have shown pupillary disturbances, 76 per cent. a Romberg sign, and 52 per cent. an absence of knee jerks. Seventy-six per cent. have given positive blood Wassermann reactions, 76 per cent. positive spinal fluid Wassermann reactions, and 76 per cent. have shown an increase in globulin. Three cases gave positive spinal fluid Wassermann reactions and negative blood Wassermann reactions, and two cases showed the contrary condition.

RESULTS OF TREATMENT

It cannot be expected of any form of therapy that it will undo destructive changes which have already occurred. But this form of treatment may be expected to control associated meningeal inflammations. The control of an associated meningeal involvement is the best explanation we can give for the improvement which these cases have shown.

We have observed no instances among well defined tabetic cases where absent knee jerks have returned, or where sluggish pupils have regained their activity. We have been impressed by the frequent improvement in the ataxia of these cases as manifested by increased steadiness in gait and a decrease in the intensity of the Romberg sign. These cases almost uniformly show improvement in nutrition, several of them showing increases in weight varying from 16 to 31 pounds.

The most gratifying results from the treatment have been shown by its effect on the subjective symptoms. In spite of the fact that the intraspinal treatments often cause severe reactions we have found these patients more anxious to continue their treatments and to return to the hospital than any other class.

From this point of view, also, it is not usual to see a return to the condition of normality, but we have been struck in questioning these patients with the fact that in most of the cases there has been a very marked and sustained diminution in the amount of discomfort from which they were suffering before the institution of treatment. In almost every one of the cases here recorded there has been relief to a great extent from pain. Similarly there has been a frequent improvement in bladder function. Of six patients suffering from vertigo, in four the condition has disappeared under treatment, one has shown improvement, and in the sixth, the condition has become progressively worse in spite of vigorous treatment.

The influence of the treatment on the serology and cytology of the disease can best be seen by reference to the table. Here it will be seen that the blood Wassermann reaction has either been reduced in degree or changed to a negative reaction in all but two cases, in which it was uninfluenced. The spinal fluid Wassermann reaction has been reduced in degree or changed to a negative reaction in nine instances, has been uninfluenced in five cases, and in one case has become stronger during treatment. The globulin reaction has been reduced in thirteen cases, and has become stronger in five. Since the introduction of the colloidal gold reaction as a routine test in cases of nervous disease eight of the cases of this series have been tested by this means on each of their admissions. Two cases have shown positive gold curves in the absence of either a positive blood or spinal fluid Wassermann

reaction. One case has shown a positive curve with a negative spinal fluid Wassermann reaction, but a positive blood Wassermann reaction. All of the cases with positive curves have shown increases in the spinal fluid cell count and globulin content. Seven of the cases have shown a decrease in the intensity of the reaction after treatment. In the eighth case the curve assumed the paretic type during treatment and the patient developed general paresis clinically (Case 20).

As we have noted above, the majority of the cases were of the tabetic type. Two of the cases were spastic in type. One of these showed marked improvement after fifteen treatments with the disappearance of spasticity on the affected side, and a return of the reflexes to normal (Case 15). In another similar case of longer duration the patient has shown moderate improvement after fourteen treatments, and can walk with the use of a cane (Case 17). We have not undertaken the treatment of any paretic cases, but have had two cases develop paresis during or after treatment. One of these patients had received six treatments over a period of four months, the other had been under treatment for tabes for twenty-four months and had received seventeen treatments (Cases 20 and 21).

We have endeavored in the accompanying table to state graphically the essential data concerning the cases, using the plus sign to indicate the presence of a condition, and the zero sign to denote its absence, and using one, two, three, or four plus signs according to the relative intensity of the symptom.

We have selected the following group of cases to discuss in greater detail:

ILLUSTRATIVE CASES

CASE 3.—History.—F. S., a man, aged 48, had symptoms during a period of three years. He has had attacks of severe shooting pains in legs at irregular intervals, with difficulty in walking in the dark.

Physical Examination.—This revealed unimpaired sensations, equal but sluggish pupils, absent knee jerks, slight ataxia of legs, and positive Romberg sign.

Laboratory Findings.—The blood Wassermann reaction was positive; spinal fluid Wassermann reaction, positive; cell count, 4.

Treatment.—This patient had received one or two intravenous injections of arsphenamin before admission to the hospital. After admission he received two injections intravenously of 0.9 gm. of neoarsphenamin and two intraspinal injections of 40 per cent. serum. After the second injection he developed the severe reaction described in the beginning of this article. He was discharged from the hospital in March, 1913, and went to Alabama to live.

Results.—Three years later in reply to our inquiry his physician, Dr. F. G. DuBose of Selma, Ala., writes: "The patient has had six intravenous injections of arsphenamin since his discharge and injections of bichlorid of mercury in the intervals. These were given in the first year after leaving the hospital. Four blood Wassermann reactions in the past year have been negative. For nine months after leaving the hospital he suffered at intervals with pains in the calves of the legs. At the present time he is free from pain or other tabetic symptoms. No Romberg's sign; knee jerks are slightly exaggerated."

Case No.	On Admission		Colloidal Gold		After Treatment	
	1	2	1	2	1	2
A B = Argyll Robertson.	13	2	2	1	1	1
† Vertigo cleared up under treatment.	14	1	2	3	1	1
† Headache improved under treatment.	15	0.5	3	3	1.5	1.5
† Transitory attacks of blindness cured.	16	2	1.5	3	1.5	1.5
† Vertigo and diplopia cured.	17	1	2	3	2.5	1.5
† Headache and diplopia cured.	18	1	2	3	2.5	1.5
† Vertigo and headache improved.	19	1	2	3	2	2
† Became parietic.	20	1	1	3	3	3

|| Became paretic.

The foregoing case is interesting because of the extremely severe reaction to one of the treatments and the improvement which occurred later, notwithstanding this reaction.

CASE 4.—History.—M. S., a woman, aged 40. Duration of symptoms twelve years. She has had attacks about twice a month of sharp shooting pains in lower part of back, radiating down sides of abdomen and into legs. Pains associated with nausea and vomiting. Recently gait has been so unsteady that she has been confined to her bed. She has frequent incontinence.

Physical Examination.—This showed a thin, pale, emaciated woman, legs wasted and thin, muscles relaxed. The right pupil was smaller than the left, neither pupil reacted. Knee jerks absent; area of diminished sensation in patch on anterior surface of right thigh, on lower anterior surface of left thigh, and on anterior surface of left leg.

Laboratory Findings on Admission.—Blood Wassermann reaction, negative; spinal fluid Wassermann, ++; cell count, 9; globulin test, positive.

Treatment.—She received four combined treatments in six weeks. At the end of this time her blood Wassermann reaction remained negative; the spinal fluid showed a negative Wassermann reaction, negative globulin test, and a cell count of 3.

Results.—This patient improved steadily after the institution of treatment. Interviewed in May, 1916, eleven months after her discharge from the hospital, she had gained much weight, and had partially resumed her housework. Pains still occurred but at much longer intervals, were of less severity, and unassociated with nausea or vomiting. Her bladder control was normal, and her gait, while still ataxic, was much improved, as evidenced by the resumption of her household duties. In reply to a letter of recent date, three years after the last interview, she stated that the improvement noted then had been sustained.

CASE 13.—History.—J. G., a man, aged 45, had symptoms that dated back three years. He complained of severe shooting pains in back and legs, unsteady gait, poor bladder control, and loss of sexual power.

Physical Examination.—Pupils equal, but react sluggishly; gait ataxic; marked Romberg sign; has to walk with two canes.

Laboratory Findings.—The blood and spinal fluid Wassermann reactions were negative; globulin test in spinal fluid, negative; cell count, 7.

Treatment.—This patient has been under treatment for thirty-eight months, and has received twenty-nine combined intravenous and intraspinal treatments. On his last admission in March of this year, the patient reported that he had had very little pain in the last three years; his sexual power had been normal for the past nine months; his bladder gave him very little trouble, and he had gained 31 pounds since beginning treatment. On admission his gait had been so ataxic that he had difficulty in walking with two canes without assistance. At present he uses only one cane, and can walk for a short distance without it.

The foregoing case has shown negative serologic tests throughout. Some observers are of the opinion that intraspinal therapy should be resorted to only in the presence of positive findings in the spinal fluid. We doubt very much if the same results could have been accomplished in this case by intravenous therapy alone.

CASE 15.—History.—W. B., a man, aged 29, complained of attack of unconsciousness one month before admission with subsequent transitory attacks of blindness; frequent attacks of vertigo and almost constant headache; weakness of the right leg. He had had a chancre and rash five years previously.

Physical Examination.—This revealed normal pupils with a right lateral nystagmus; convergence normal. All deep reflexes exaggerated on the right; ankle clonus and positive Babinski obtained on the right; Romberg positive; spastic gait with dragging of right leg.

Laboratory Findings.—The blood Wassermann reaction was + + +; spinal fluid Wassermann, + + +; cells, 119; globulin test, + +; colloidal gold curve, 05 1 15 2 3 15 15 0 0 0.

Treatment.—This case received twelve injections of mercury at the time of the secondary eruption. On admission the patient was put on mixed treatment and was given two intravenous injections of arsphenamin, 0.6 gm. each, and two intraspinal injections of concentrated arsphenaminized serum at intervals of two weeks. He was then treated as an outpatient, being admitted to the hospital once a month for intraspinal injections—fifteen in all. During the intervals he was given mixed treatment.

Results.—At the end of fifteen months this patient is now pursuing his trade as an electrician. The subjective symptoms have entirely disappeared; gait and reflexes are normal, and the serologic findings are negative. Monthly blood and spinal fluid Wassermann tests have been negative for the last five months. He has gained weight under treatment.

CASE 17.—History.—E. S., a woman, aged 39, complained of gradually increasing weakness of the right arm and leg, diplopia, generalized muscular pains and vertigo; difficulty in starting micturition. Duration two years. Has been married twice; no definite history of infection, although the patient believes her first husband had syphilis. One child living and well—later, one miscarriage.

Physical Examination.—This revealed normal pupils, right external strabismus; right knee jerk absent, left exaggerated; Romberg sign positive; gait spastic, bilateral Babinski reflex; no disturbance of sensations.

Laboratory Findings.—The blood and spinal fluid Wassermann reactions were negative; cell count, 24; globulin test, positive; colloidal gold curve, 1 2 3 2 2 0 0 0 0 0.

Treatment.—The patient remained in the hospital two months. During that time she received mixed treatment and two combined intravenous and intraspinal injections. She is now being treated as an outpatient, having received a total of fourteen treatments in twenty-one months.

Results.—At the latest visit she reported marked improvement with a returning use of right arm and leg. She does light housework. Gait still spastic; there is marked improvement in pain, vertigo, and bladder control. Has lost 10 pounds in weight. No strabismus; knee jerks and Romberg sign unchanged. Spinal fluid cells and globulin reaction are now normal, and colloidal gold curve is improved.

CASE 20.—History.—C. V., a man, aged 37, had symptoms during a period of ten years. He has had frequent attacks of pains in legs, and severe attacks of vomiting, unassociated with pain. Legs weak and gait unsteady. Vision in the left eye was poor.

Physical Examination.—This revealed Argyll Robertson pupils, absence of knee jerks, a moderate Romberg sign, well marked ataxia of lower extremities, and slight ataxia of hands.

Laboratory Findings on Admission.—The blood Wassermann reaction was doubtful, probably positive; spinal fluid Wassermann, negative; cell count, 30; globulin reaction, positive.

Treatment.—This patient received seventeen combined treatments in two years. Under the treatment his pains became less frequent and less severe in character and there was apparent improvement in his gait. A month before his last admission he began to develop delusions of grandeur, and on admission he was unmistakably parietic and had to be transferred to another institution.

The foregoing case is interesting in that treatment was begun at least a year and a half before the patient became of parietic type and that paresis developed in spite of the treatment.

CONCLUSIONS

In this series of cases 214 intraspinal treatments have been given. Severe reactions have occurred, particularly in Case 3, but we have seen no permanent injury or ill effects result from the intraspinal use of auto-arsphenaminized serum.

The treatment has uniformly been of benefit in its effect of increasing the patients' comfort by lessening the severity and frequency of their pains, in frequently improving bladder control, and in improving their nutrition. Improvement in station and in gait has frequently been observed in these cases, and in some, to a very marked extent.

PERNICIOUS ANEMIA WITH MENTAL SYMPTOMS

OBSERVATIONS ON THE VARYING EXTENT AND PROBABLE DURATION OF
CENTRAL NERVOUS SYSTEM LESIONS IN FOUR
NECROPSIED CASES *

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CINCINNATI

Our knowledge of pernicious anemia is being constantly enriched, especially our knowledge relating to the changes occurring in the nervous system. And justly so, because after all, next to the blood changes, the most characteristic and frequent clinical findings are on the part of the central nervous system. Minnich† (1893) demonstrated lesions in the spinal cord in approximately 70 per cent. of cases of pernicious anemia. Of forty-one cases treated by Billings (1900), forty showed neurological symptoms. In a very recent (1919) report of the 150 moderately advanced cases of pernicious anemia examined at the Mayo Clinic, Woltman found indisputable evidence of nervous tissue disintegration in over 80 per cent.

Formerly the neuropathology of this disease consisted chiefly in descriptions of the changes taking place in the spinal cord. It has been but comparatively recently that investigation has shown that these changes are not limited to the cord, but involve other parts of the central nervous system as well. (Pfeiffer, Barrett, Woltman.)

ETIOLOGIC FACTORS

In discussing the etiologic relationship between the brain and cord changes on the one hand and the blood changes on the other, one is confronted by various theories, each of which has its ardent supporters. The reason for this diversity of opinion is our ignorance of the fundamental cause or causes underlying this disease. Practically all are agreed that the clinical syndrome which we call pernicious anemia is produced by one toxin, the nature and origin of which, however, are still mooted points.

We feel safe in assuming that the disease is due to a toxin because of experimental work performed along similar lines in cases of anemia

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†Bibliography will be found at the end of the article.

due to the *Bothriocephalus latus*. It was long known that all individuals harboring these parasites did not develop pernicious anemia. Of those that did develop it nothing was disclosed either during life or at necropsy to show that they possessed any characteristics that might be regarded as predisposing them to this disease. The natural inference, then, was that the parasite was the variable factor. In other words, some change occurred which transformed the parasite from a harmless, though unwelcome, inhabitant of the intestinal tract into a deadly menace.

Elaborating on this idea, Shapiro advanced the theory that when the worm died or became diseased, noxious substances were produced which on being absorbed produced the anemia. The work of Wilt-schur strongly supports this view. He examined twelve worms, the removal of which cured severe anemias and found that in "all cases the worms were either dead, decomposed, or sick."

Reasoning by analogy, we feel confident that the Addison-Biermer type of anemia is also due to a toxin. As to its origin, we as yet know nothing very definite. Much work has been done in this connection. The regularity with which changes occur in the gastro-intestinal tract, has led many to believe that this condition is essentially a disease of the gastro-intestinal tract (Grawitz-W. Hunter). Atrophy of the gastric mucosa, achylia gastrica, and intestinal stasis are frequently encountered. Berger and Tsuchiya found that extracts of the gastric and intestinal mucous membranes from patients dead of this anemia were more hemolytic than extracts of normal mucous membranes. This statement, however, is disputed by Ewald and Freidberger.

It is well known that certain chemical substances, such as oleic acid, saponin, phenylhydrazin, b-amino-azolyl-ethylbenzaldehyd and p-oxyphenylethylamin are capable of producing intense hemolysis. One of these substances, namely p-oxyphenylethylamin, has been isolated by Iwao from autolyzing pancreas, putrefying horse flesh and from Swiss cheese. Conceivably this compound may arise in the intestinal tract as the result of decomposition of food. Berthelot and Bertrand have shown that among the flora of the intestinal tract there is a bacillus, the *B. aminophilus*—which is capable of producing p-oxyphenylethylamin from tyrosin. Thus we can readily see how intestinal stasis could be a great factor in the production of pernicious anemia.

On the other hand, others have taken the stand that the increased production of hemolysins is due to a condition of hypersplenism. The toxin is carried by the blood to the spleen where certain changes occur. According to Moffit, "erythrolysis does not take place in the spleen but in some way the erythrocytes are sensitized and prepared for later destruction in the liver, marrow or lymph glands." From this view-

point it would be easy to account for the beneficial results that often follow splenectomy.

Granting that a toxin is responsible for this condition the lack of the fundamental knowledge as to its origin and *modus operandi*, makes it all the more difficult in trying to solve the causal relationship between the blood and the central nervous system changes. We have a mass of clinical and histopathologic data that has been accumulating ever since Addison first described pernicious anemia in 1855. Investigators have naturally tried to correlate these facts, and in correlating them, have advanced many theories. Most of these theories have long since fallen by the wayside; others, however, have remained and can be briefly summarized as follows:

1. A toxin produces the anemia which in turn produces another toxin that causes the changes in the brain and spinal cord.
2. The anemia itself acts as a toxin which through malnutrition causes the changes observed in the central nervous system.
3. The toxin acts independently on the blood and on the central nervous system.

Before discussing the relative merits of these theories it is essential to bear in mind certain well established facts. The absolute lack of correspondence between the brain and cord changes on the one hand and the anemia on the other hand both from anatomic and clinical standpoints has long been known.

Clinically there are many cases in which the neurological symptoms appear before the anemia. In other cases, both conditions arise simultaneously, and in still other cases, the anemia is present long before the appearance of any neurological disturbances.

Similarly, either condition may improve without a corresponding improvement in the other, or both may show a coordinate improvement.

Furthermore, it has been stated (Goebel) that cases that clinically show disturbances of the central nervous system may on section show no demonstrable organic changes.

From the above statements, it is obvious that the first two theories are untenable. So long as it is possible for the nervous symptoms to precede the appearance of the anemia, then it is impossible to maintain that the anemia either directly or indirectly, that is, through the production of another toxin, produces the cord and brain changes.

The third theory, however, overcomes this objection. If we assume that the toxin acts independently on the central nervous system and on the blood, then we can say that in one instance, the toxin acts first on the central nervous system and then on the blood, thus producing those cases in which the disease is ushered in by the nervous symptoms. In another instance, it acts on the blood first. These are the cases in

which the anemia precedes the development of neurological symptoms. In still another instance, the toxin acts simultaneously on the central nervous system and on the blood, thereby giving rise to a clinical picture showing both brain and blood changes at once. (Nonne-Billings.)

So far so good. But, on the basis of this theory, how can we explain the fact that in cases where similar therapeutic measures, for example, splenectomy, have been instituted, some will show an improvement in the neurological symptoms without any improvement in the blood picture, while others, reversely, will show a betterment in the anemia without a corresponding betterment in the cord and brain changes.

Decastello* performed a splenectomy on a patient who had suffered from the disease for less than a year. The anemia was severe and the spleen but slightly enlarged. Since the operation, the patient has shown marked improvement clinically but with no alteration in the blood picture.

Harpole,* on the other hand, performed a splenectomy on a patient who had been known to have had the disease for at least two years. The anemia was moderately severe. Following the operation there was an immediate improvement. The patient has continued in fair health with only a slight anemia but with *persistence of the spinal cord symptoms*.

Furthermore, how will this theory account for those cases which showed clinical evidence of involvement of the central nervous system, but in which the pathologist, on necropsy, is unable to discover any demonstrable lesion?

It seems to me that if we judiciously combine the second and third theories into some such theory as the following, we shall have one that will account for all of the various manifestations of pernicious anemia. Briefly stated it would read as follows:

1. One toxin produces pernicious anemia.
2. This toxin acts independently on the blood and on the central nervous system.
3. The anemia itself, after it has persisted for a considerable length of time, interferes with the metabolism of the nerve cells, thereby indirectly enhancing the poisonous action of the toxin so that instead of being merely an irritant to the nervous tissue, it becomes a highly destructive agent. In this way, the changes which at first were purely temporary and functional now become permanent and organic.

In other words, I think that we can with justice assume that the nervous structure is open to two lines of attack, namely, (1) a direct or frontal attack by the toxin, and (2) an indirect or flank attack by

* See Pearce, Krumbhaar and Frazier: The Spleen and Anemia.

the anemia. If the anemia is improved before the latter attack has materialized we get an improvement in the neurological symptoms and such a patient on necropsy will show no pathologic cord or brain lesions.

If, however, the anemia has persisted long enough for both frontal and flank attacks to materialize, then improvement in the blood picture will not be followed by a corresponding improvement on the part of the central nervous system. Such cases coming to necropsy will show the changes that are usually found in the cords and brains of pernicious anemia patients.

This theory is in line with the views of Bonhoeffer who thinks that the psychoses that some patients present are not due to the direct action of the toxin but to the interposition of some metabolic changes in the nerve cells.

Christian's recent work strengthens this view also. He tested the renal function in fourteen cases of pernicious anemia and found that the latter produced a chronic nephritis which could be improved by improvement of the anemia, provided the latter had not persisted for too great a length of time. His conclusions are:

In patients with pernicious anemia the disease is accompanied by a disturbance of renal function, as measured by renal dietary tests, which is similar to that found in patients with advanced chronic nephritis. In these patients there is no other evidence of chronic nephritis and the disturbance appears to be due to the anemia, decreasing with the subsidence of the severity of the anemia *unless the anemia is maintained so long that a permanent disturbance of renal function ensues.* [Italics mine.]

This theory stated in its entirety would read as follows:

1. One toxin causes both the blood and the central nervous system changes.

2. This toxin acts independently on the blood and on the central nervous system. This would account for those cases in which (a) the neurological symptoms manifest themselves before the anemia, or (b) in which the anemia precedes the brain and cord changes, or (c) in which both conditions arise simultaneously.

3. As soon as the typical blood picture of pernicious anemia develops and persists for a considerable length of time, the metabolism of the nerve cells is so impaired that the changes which were purely functional at first and due to the irritating action of the toxin alone, now become organic and permanent. Hence, no matter how greatly the physical state is improved, be it through splenectomy, transfusions, or drugs, no improvement on the part of the nervous system follows. The damage is irreparable. If, however, the hemolysis has not lasted very long, then improvement in the neurological symptoms may be expected to follow the exhibition of proper therapeutic measures. We can thus

account for the apparent contradictory results in the cases reported by Decastello and Harpole. In the latter case, the anemia had lasted for at least two years. Removal of the spleen was followed by an improvement in the blood picture but with no improvement on the part of the central nervous system. Here we can rightly assume that the anemia had interfered to such an extent with the metabolism of the nervous elements that permanent lesions had been produced.

In the former case, the anemia had existed for a much shorter period and therefore splenectomy was followed by improvement in the neurological symptoms.

In line with this theory is the fact brought out by Pearce, Krumbhaar and Frazier and their co-workers, that if splenectomy is performed before the blood has reached an extreme degree of deterioration, not only is the operative risk lessened but the improvement is greater and more lasting.

Finally, on the basis of this theory it is easy to account for those cases which during life showed clinical evidence of involvement of the central nervous system but which on necropsy showed no lesion, either in the cord or brain. In these cases, the irritant action of the toxin had not been aided by the anemia.

MENTAL SYMPTOMS OF PERNICIOUS ANEMIA

Many observers have called attention to the mental symptoms that frequently occur in pernicious anemia patients. The early investigators simply noted the somnolence, apathy, and coma that usually preceded the fatal termination of the disease. Later, however, it was noted that the mental symptoms were not simply terminal but that in a great many cases they constituted a dominant part in the clinical picture. Marcus and Langdon have each reported cases in which the mental changes appeared before the anemia or, at least, before the blood picture was sufficiently developed to warrant the diagnosis of pernicious anemia. Langdon termed these cases "prepernicious anemia." The psychic disturbances may range from mild depression to violent maniacal outbursts. Some cases will show irritability, hyperkinesis, delusions and hallucinations. Other cases will show indifference, apathy, and severe melancholia. Whenever a distinct psychosis, such as manic-depressive insanity, is present, the majority of observers look on it as separate and distinct from the pernicious anemia. At most, in such cases the pernicious anemia may be regarded as a predisposing factor. At present it is generally accepted that the psychotic manifestations should be classified with the exhaustion and toxic-infectious psychoses. Barrett expressed it very clearly when he said:

As to the clinical position, it would seem that they must be placed among the paranoid conditions which are symptomatic of a toxic organic process affecting the central nervous system—analogueous to the paranoid conditions which have been noted in tabes, alcoholism and from certain drugs.

It has been but comparatively recently, however, that efforts have been made to link up the mental disturbances with the cortical changes. Barrett found many pathologic changes in the cortex but as a whole, not of the specific type. They appeared to be rather similar to those changes which occur in conditions of chronic intoxication, due, for example, to chronic alcoholism. The blood vessels showed the most constant changes. These were in the nature of swelling of the intimal cells and in some active proliferative changes. The most important findings, however, are the focal lesions, which correspond very closely to the lesions so characteristically present in the cord.

Woltman made a very thorough and exhaustive study of seven cases. His findings led him to the conclusion that the brain and cord changes run fairly parallel and with about the same frequency; and, furthermore, that patients who show degenerative changes in the spinal cord at necropsy, usually show the same type of lesion in the brain also. In the medullary substance of sections of different levels of the brain, he also found areas of degeneration of the Lichtheim type that are identical with those that are usually found in the posterior and lateral columns of the spinal cord. In addition to these focal areas of degeneration he noticed diffuse areas of degeneration in the long association tracts and in the short commissural fibers that pass from one gyrus to another. He calls attention to the fact that the gray matter shows involvement of a focal nature also, the cells of the marginal gray layer being principally involved.

REPORT OF CASES

Technic.—The following report is based on the study of four cases. In each case the brain and spinal cord had been fixed in formaldehyd solution and cut in the frontal plane. Blocks of tissue about 5 mm. in thickness were taken from each of the following regions: (1) left motor area; (2) middle of pons; (3) middle of medulla; (4) cervical region of cord; (5) dorsal region of cord; (6) lumbar region of cord. In addition, sections were also taken in some cases from the right motor area, the left internal capsule, and the peduncles. These were selected because, macroscopically they seemed to offer promising material for study. These blocks were then mordanted, embedded in parlodion, cut, under alcohol, into sections 25 microns in thickness and stained by the Weigert method. Those stained with cresylecht-violet, had formalin fixation and paraffin embedding and were cut into sections 6 microns in thickness. Some of the sections were also stained with hematoxylin and eosin.

CASE 1 (Necropsy 15-106).—*History.*—O. D., a white man, aged 75, was admitted to the psychopathic department, Boston State Hospital, Sept. 27, 1915. He was mentally confused and had hallucinations and delusions of various kinds.

Family History.—The family history is entirely negative. There is no history of nervous or mental diseases in any of the collaterals.

Personal History.—The patient was born in Germany and had the ordinary diseases of childhood. He had a severe attack of rheumatism sometime between the ages of 50 and 60 years. About four or five years ago he began to have bladder difficulties. He left school at the age of 15 and spent the next twenty-six years at sea. He then came to this country and worked steadily for a ship concern until the onset of the present illness, which prevented him from performing his duties properly. His daughter stated that he left his work because his mind gave out and because he had dizzy spells and would fall on the street. His condition gradually became worse and he had begun to dislike people, preferred to stay alone, was irritable and ugly. He was always complaining and found fault with everything. He had delusions of persecution. His daughter had become afraid of him because of his ugliness. For the past three years he had been very pale and complained of numbness of the feet. However, he did not drag his feet.

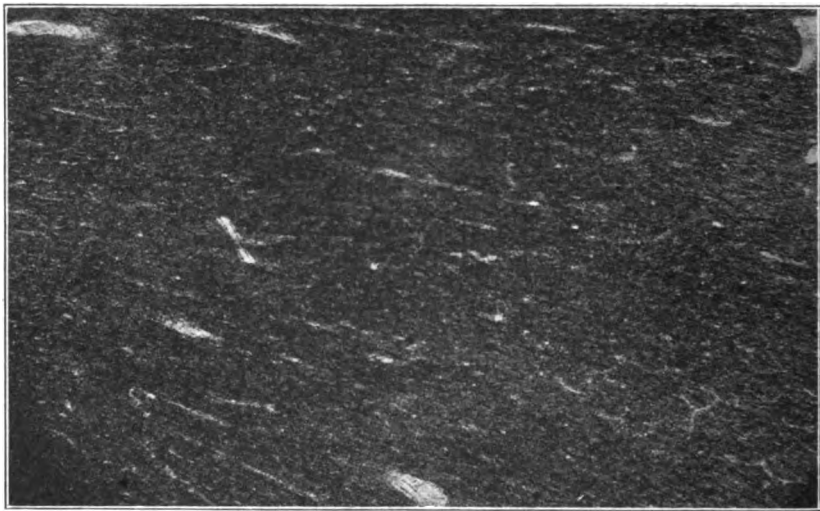


Fig. 1 (Case 1).—Section of left motor area, showing areas of degeneration in close proximity to the blood vessels. One, in the upper right hand corner, is at the point of bifurcation of a capillary. In the upper left hand corner a focus of degeneration is seen surrounding blood vessels. Weigert's myelin sheath stain. Magnified about 40 diameters.

Ten weeks before admission, the patient fainted and afterward had a chill, a period of vomiting and later, fever which reached 102 F., after which he had a peculiar cold period. It seems that the mental confusion, hallucinations, rambling and incoherent talk have chiefly developed since the onset of this attack of chill, vomiting and fever. Previously he had always been constipated but during this bed-ridden period there was diarrhea and he had no control of rectum or bladder.

His hallucinations varied. At one time he saw a man in the room with him, sitting with his hat on; at another time, he saw trucks and a steamer in his

room. At times he feared that he was going to be put down into the cellar, and at other times he thought he was being kept there. He also had periods of memory defect.

Examination.—On examination, the face was seen to be puffy and pasty and the skin unduly pale. There was a systolic murmur at the apex which was feebly transmitted to the axilla. The aortic second sound equaled the pulmonic second sound. The pulse was regular and of good volume. The blood pressure was: systolic, 130; diastolic, 70. The breath sounds were faint and crepitant; râles were heard all over the back and front. The liver was enlarged but not tender. The spleen was not palpable.

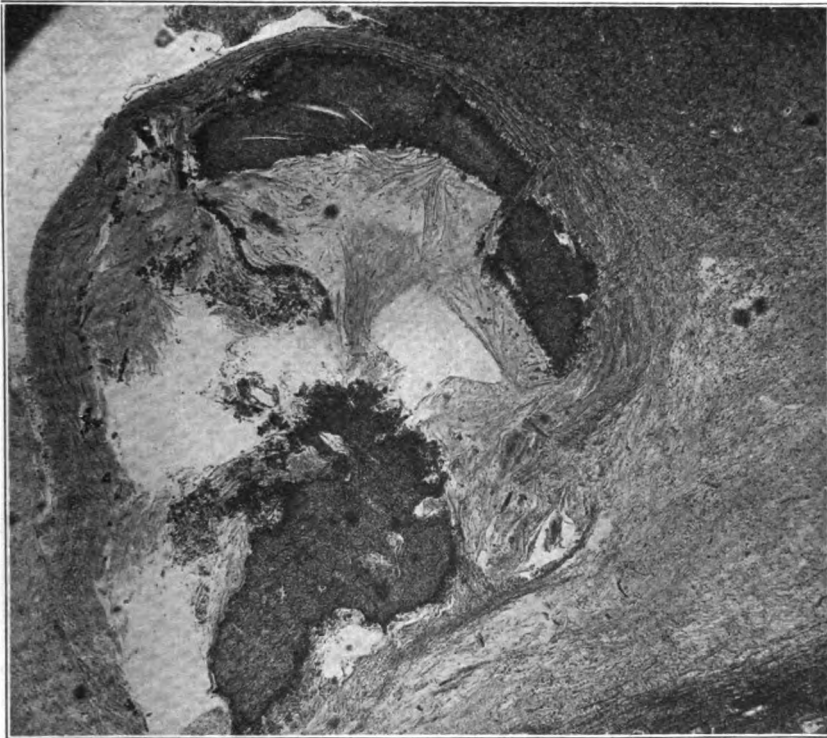


Fig. 2 (Case 1).—A large area of softening in the cortex produced by rupture of a large blood vessel. Weigert's myelin sheath stain.

Neurologic examination showed normal reacting pupils. The fundi could not be examined on account of the patient's restlessness. The triceps and biceps were present and equal on both sides. The abdominal reflexes were only obtained in the left inguinal region. Both knee jerks were absent. The same was true of the Achilles reflex. There were no Babinski, Gordon or Oppenheim reflexes. There was no ankle clonus. The deep sensibilities were not tested because the patient could not cooperate.

The urine showed a slight trace of albumin and an occasional red blood corpuscle, and a few granular casts and a rare hyaline cast. The blood Wassermann reaction was negative. Examination of the spinal fluid showed the

following: Fluid, clear; albumin, +++; globulin, ++; cells, 4; small lymphocytes, 100 per cent. Colloidal gold chlorid test, 0000000000. Blood Examination: This revealed the following: Hemoglobin, 30 per cent. (Sahli) with a color index of 1.66; red blood cells, 2,528,000; white blood cells, 4,600. A differential count (100 cells) gave polymorphonuclear leukocytes, 71 per cent.; small lymphocytes, 16 per cent.; large lymphocytes, 10 per cent.; eosinophils, 3 per cent.

There is a great variation in the size of the red blood cells with a large percentage of macrocytes. Poikilocytosis is marked. No achromia.

Course.—September 28: Patient has been lying quietly all day in a semicomatose condition. He does not comprehend questions, rarely speaks, and when he does, cannot answer questions relevantly. His mind wanders deliriously, calling for "Annie" and telling fabulous tales.



Fig. 3 (Case 1).—A so-called Lichtheim plaque in the left internal capsule. Weigert's myelin sheath stain.

September 30: Condition the same. The skin is very pale and has a slight lemon yellow tint. Both knee jerks and Achilles are absent. All the toes were drawn up on stroking the sole of the foot and in testing for Oppenheim's sign.

October 1. The patient has continued in the same low grade semiconscious semidelirious condition previously noted. He continues to call deliriously for "Annie," but otherwise makes practically no intelligent remarks, occasionally rambling about "the boat." The yellowish tint of the skin is more marked. Moist, bubbling râles could be heard all over the chest. Patient failed rapidly and died early in the afternoon.

Necropsy.—This was performed six hours after death. The body is that of a well built and nourished, white male, 176 cm. in length. The skin is waxy

gray with a slight yellowish cast. There is a faint edema of the lower legs and some atrophy of the left thigh. The pupils measure 5 mm. in diameter and are equal. There is a superficial decubitus over the sacrum. Rigor mortis is faintly present.



Fig. 4 (Case 1).—Here the miliary foci described by Preobrajensky are very conspicuous. The contrast between them and the larger so-called Lichtheim foci, three of which are also shown in this picture, is very striking. The foci of Preobrajensky are much smaller, more numerous and more sharply defined than the Lichtheim plaques. Although not shown in this photograph, in a good many cases an undamaged nerve fibril may be seen traversing a small focus. Weigert's myelin sheath stain of pons.

Ventral Section: The fat is lemon yellow in color and measures 2 cm. over abdomen and 1 cm. over thorax. The spleen is adherent to the external lateral surface. The appendix above the pelvic brim measures 7 cm. in length. The bladder is distended and the intestines somewhat injected. The diaphragm arches to the fourth rib on the right side and to the fifth interspace on the left side. The gallbladder is distended and contains many stones.

Thorax: The bone marrow is yellowish pink in the sternum. There is no free fluid in either pleural cavity, but there are adhesions at the apex of the right lung. The pericardium is heavily loaded with fat. The apex of the heart is in the fifth interspace.

Heart: Weight, 453 gm. The epicardial fat is fairly abundant. The descending branch of the left coronary shows sclerosis. The right coronary and circumflex arteries show constrictions with calcifications. Every valve is



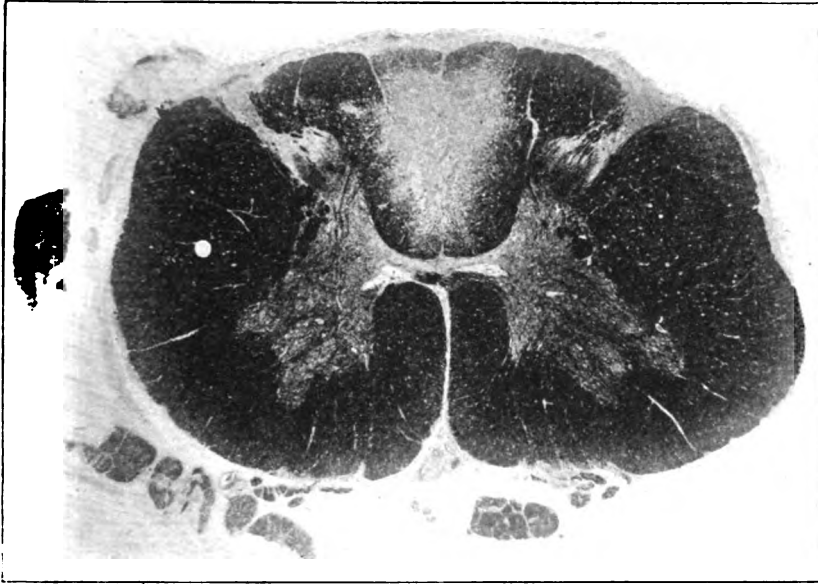
Fig. 5 (Case 1).—Another view of a different section of the pons. The relationship of the large plaques of degeneration to the blood vessels is very clearly shown. Here we see again the formation of an area of degeneration at the bifurcation of a capillary. A large number of the smaller foci are also present. (Weigert method.)

thickened, particularly the aortic which shows distortion of the cusps. There are some vegetations which are calcified around the origin of the left coronary in the internal surface of the aorta. The myocardium is pinkish gray in color and contains multiple white streaks measuring from 0.5 to 0.6 cm. in extent.

Lungs: Weight—left lung, 385 gm.; right, 1,200 gm. The right lung pits on pressure, but is crepitant for the most part. There is a slight thickening

of the pleura at the apex. The bronchi are reddened and show frothy fluid adherent to mucus.

The left lung has a collapsed area in the lowest part of the upper lobe and in the posterior part of the lower lobe. Section of this shows it to be somewhat redder but not wetter than usual. The bronchi are reddened but the peribronchial lymph nodes are not enlarged.



Figs. 6, 7, 8 (Case 1).—Sections of the cervical, dorsal and lumbar regions of the spinal cord. The degeneration of the posterior columns is marked. This degeneration is only moderately severe in the lumbar region, but becomes progressively worse in the upper portions of the cord. It reaches its climax in the cervical region. Here, the destruction is seen to be very severe. Within the area of degeneration, large, jagged holes are present. This is in marked contrast to *tubes dorsalis*. In the latter condition, the degeneration of the posterior columns, as a rule, is greater in the lumbar region than in the cervical region. The degeneration in the lateral columns, which as a whole is much less than in the posterior columns, increases in intensity from above downward. The posterior roots in the lumbar section show evidences of degeneration. The hole seen in the lateral column of each section has been made to mark the right side of the cord. This applies to all the following photographs of the cord. Weigert's myelin sheath stain ($\times 10$).

Spleen: Weight, 165 gm. There are two fetal lobulations on the lower border. The capsule is somewhat thickened and wrinkled. The pulp is red and watery, and retracts on section. The trabeculae are increased and the malpighian bodies are numerous.

Adrenals: They are embedded in fat and are large. On section they show marked mottling of cortex and medulla with yellow and red. The medulla is scarcely to be differentiated from the cortex.

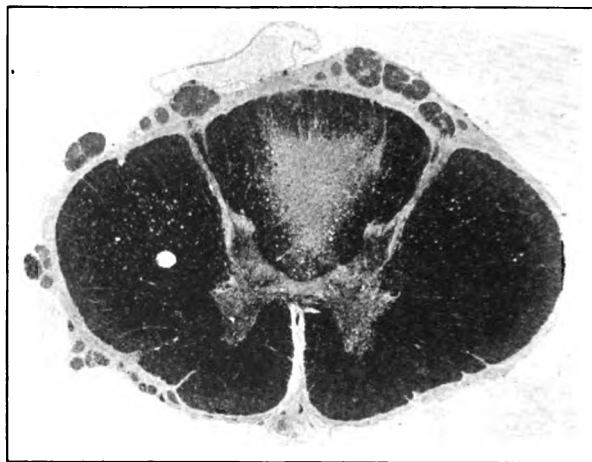


Figure 7

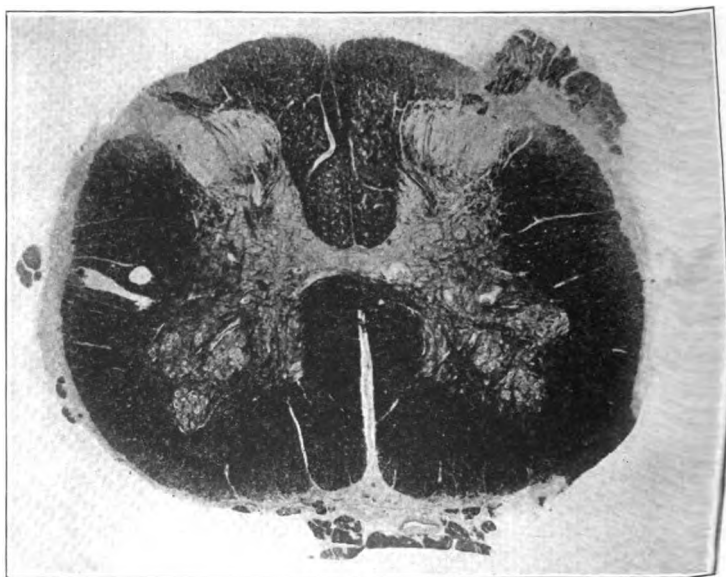


Figure 8

Kidneys: These are deeply embedded in fat. The capsule strips with difficulty.

Liver: Weight, 1,800 gm., and has a yellowish pink color. The capsule is slightly thickened and there is a focal area of adhesion to the diaphragm. Section shows a fairly pale homogeneous substance. The gallbladder contains forty-eight stones.

Pancreas: The splenic artery is tortuous, markedly sclerosed and calcified. Every level of the pancreas examined shows fat replacement in varying amounts.

Gastro-Intestinal Tract: Section of stomach shows the wall somewhat thickened and glossy. No rugae are present. Yellow mucus drips from the surface. There is nothing of note in the intestinal tract.

Special Examination: The bone marrow of the left femur was inspected, and found to be raspberry red in color.

Brain: The dura is very adherent, but not particularly thickened. The superior surface of the brain generally is firmer than normal. The convolitional pattern is rich. The pia mater is not thickened except slightly in the sulci over the vertex.

Base of brain shows a small aneurysm in the middle part of the left posterior communicating artery. The fourth ventricle shows clear granulations. The brain weight is 1,100 gm. which, according to Tigges' formula, shows a loss of 308 gm. The cord shows minute specks of translucency in the middle of the posterior columns.

COLLOIDAL GOLD CHLORID REACTION

	1	2	3	4	5	6	7	8	9	10	
Cortex	1	2	2	2	2	2	1	1	0	0	Bloody
Right base.....	1	2	1	1	1	1	0	0	0	0	Bloody
Left base.....	3	2	2	2	1	1	0	0	0	0	Bloody
Third ventricle *.....	0	0	0	0	0	0	0	0	0	0	
Spinal fluid.....	0	0	0	0	0	0	0	0	0	0	
Pericardial fluid.....	0	0	0	0	1	1	1	2	2	3	

Histologic Examination.—Weigert Sections—Left Motor Area: Macroscopically, one sees a cross-section of a very large blood vessel with thickened walls. Surrounding it, in the medullary substance, is a very large area of softening which shades off gradually into the surrounding normal tissue. On microscopic examination, this area of softening is seen to be composed of destroyed nerve fibers, with here and there, small patches of neuroglia and intact nerve fibers. The entire part appears cribriform owing to the presence of many large vacuoles some of which have apparently run together to form large cavity-like spaces. In the medullary portion, the blood vessels are thickened, the perivascular spaces are dilated and in a great many instances there is a thinning out of the myelin sheaths in their immediate neighborhood. In many cases, this destructive action has gone on to complete degeneration so that irregularly circumscribed areas which vary in size and structure have been formed. Some are filled with debris and crossed by a few undegenerated nerve fibers, while others are clear and hyaline-like in appearance. At the bifurcation of one of the capillaries there is a so-called Lichtheim focus. In the cortical area the blood vessels are numerous and have very thick walls. Some are seen to be ruptured. The perivascular spaces are greatly distended.

Internal Capsule: Here there are also vascular changes both in the gray and white matter. Many plaques of different sizes and shapes are seen sur-

rounding the blood vessels. The perivascular spaces are uniformly distended. In a small portion of the medullary substance one can see a few of the miliary foci described by Preobrajensky.

Pons: The most striking feature in this section are the miliary foci of Preobrajensky. They are very numerous and confined entirely to the medullary portions and are most numerous in the center. Practically all are of uniform shape but not of uniform size, some being three times as large as others. The smallest are about the size of small lymphocytes. They are fairly definitely circumscribed and have a punched out appearance. In many, an undamaged nerve fibril may be seen passing through the destroyed area. In addition to these miliary foci of Preobrajensky there are quite a number of well defined Lichtheim plaques. These bear a close relationship to the blood vessels.

Cord: With the unaided eye one can see a distinct circumscribed degeneration of the column of Goll. In the cervical region, this has proceeded to apparent cavity formation. The degeneration, although still sharply defined in the thoracic region, becomes less and less marked as we pass to the lumbar region. The reverse, however, is true of the lateral columns. Here the degeneration, which is much slighter than in the posterior columns, increases from above downward. On microscopic examination, we see that the column of Goll is practically entirely destroyed. Several large spaces with irregular jagged edges are conspicuous. Smaller vacuoles are numerous. Here and there are evidences of small hemorrhages. The blood vessels are intensely congested.

Cresylecht-Violet Sections—Left Motor Area: The pyramidal cells appear shrunken and granular. Many of the pericellular spaces are filled with a large number of satellite cells. The perivascular spaces, especially those of the cortical area, are distended.

Internal Capsule: Here and there are small islands of sclerotic tissue, apparently not related to the blood vessels. The pyramidal cells are uniformly shrunken, irregular in outline, and in a great many, the nucleus is displaced. Some satellitosis is present. From one pericellular space, the nerve cell had entirely disappeared and its place is occupied by five satellite cells.

Pons: Small, clear, oval areas corresponding in size and position to the miliary foci of Preobrajensky, seen in the Weigert section of the pons, are also present here. In the gray matter there are many small patchy areas of degeneration. The nerve cells are not as noticeably abnormal as those of the preceding sections.

Medulla: Pigmentation of the nerve cells is marked. The perivascular spaces are tremendously distended and filled with debris. Large areas of degeneration are present in the white matter. The pyramidal tracts are full of vacuoles. Two types of glia cells, differing principally in size, were observed. The larger ones, in all probability, simply represent more mature forms. They are profusely distributed over the entire section but more particularly in the region of the olivary nuclei.

Cord: The posterior columns contain numerous thin walled capillaries which are surrounded by large amounts of glia cells and fibers. The glia replacement cells are of comparatively large size and of the stellate type. An occasional undegenerated or only partially degenerated nerve fiber is seen. The increase in the number of blood vessels is most noticeable in the dorsal portion of the cord. The nerve cells show various changes. Some are very much shrunken and irregular in outline; others contain numerous variously-sized granules; still others are swollen and contain a large amount of yellow pigment. Pigmentation is present both in the anterior and posterior horn cells, but occurs

more frequently and uniformly in the former. This pigmentation differs from that normally found in the cells of persons of advanced years, in that it is diffuse, somewhat granular and not clumped at one pole of the cell body. In these cells, the pigment is centrally located and apparently has pushed the protoplasmic substance to the extreme periphery of the cell. In some of the cells, the nucleus is displaced to one side and stains poorly.

CASE 2 (Necropsy 16-59).—History.—C. C., a white woman, aged 54, was committed to the Boston State Hospital, April 25, 1916, with the diagnoses of pernicious anemia and symptomatic psychosis.

Family History.—Her mother died at the age of 50 of tuberculosis. Of six siblings, two have died of tuberculosis; one is now in a hospital suffering from tubercular trouble; one (a sister) is excessively alcoholic and has seizures with unconsciousness, and one died in infancy.

Personal History.—The patient was born at Salem, Mass., in 1862. Her education was limited but she was able to read and write. She drank a little beer and smoked cigarets for about a year. She formerly used snuff. Her sexual habits, as far as could be ascertained, were normal. She was never very cheerful, and many times greatly depressed. She was somewhat obstinate, but in many ways showed that she could be very sympathetic and kind. She was known to be hypochondriacal and apprehensive. She was a good housekeeper, was sociable and made friends. Her first marriage occurred when she was 18 years old. By that husband, she had one child, who died at the age of 3 of "water on the brain." At the age of 36, she was again married, but by this marriage she had no children and no miscarriages. Since the second marriage she has led a very lonesome life.

Medical History.—In childhood she was at the Salem Hospital because of anemia. According to the statements of her acquaintances, she has always been pale. She always claimed that she was a spiritualist and "could see dead people." Early in 1915, she went to the Eye and Ear Infirmary because she thought that she had cancer of the throat. In May of that year, she was for nearly three weeks a patient at the Massachusetts General Hospital with what was diagnosed as pernicious anemia. At that time she was irrational and had visual hallucinations. She saw animals and would point her fingers at imaginary objects. She walked unsteadily and fell frequently but never lost consciousness. Sometimes she had complete loss of vision for a few minutes. Until a year previous to this attack, the patient was able to do her housework. Six weeks ago she became much worse; very excitable, swore and used obscene language. Recently she carried on an imaginary conversation with her deceased daughter. She has also turned against her niece and niece's little girl of whom she has been very fond. On occasions she would "jump out of bed at people." There were no homicidal or suicidal tendencies.

Physical Examination.—The patient was a well developed and well nourished woman of 54. Her face was of a striking pallor with a lemon yellow tinge. The mucosae were very pale. There was a harsh systolic murmur at the apex that was transmitted upward to the axilla. The liver was enlarged but not tender. The spleen was palpable in the left hypochondrium and the dulness extended to 3 cm. above the umbilical line. The blood pressure readings were: systolic, 105; diastolic, 45.

Neurologic Examination.—The pupils were equal and regular. They reacted well to accommodation but very sluggishly to light. There was no nystagmus or strabismus. The tongue protruded in the midline and appeared very anemic. The arm reflexes were all hyperactive. There was hyperesthesia of the lower

extremities. The knee jerks were equal but sluggish; the Achilles active and equal, and the plantars hyperactive. No Babinski, Gordon or Oppenheim reflexes were obtained. There was no ankle clonus.

A formal mental examination could not be made on account of her mental state. The patient appeared to be in a dazed, half asleep state. It was difficult to get her to comprehend questions but when her attention was gained, she usually answered the questions fairly well. When asked where she was born, she delayed before answering, then asked what the question was. She finally said that she was born in Salem. She did not know how long she had lived there. She answered questions as if very weary and gave the impression that the incorrect answers were due, in part, to lack of effort. She persisted in the feeling that her niece had intended to injure her and refused to see her.

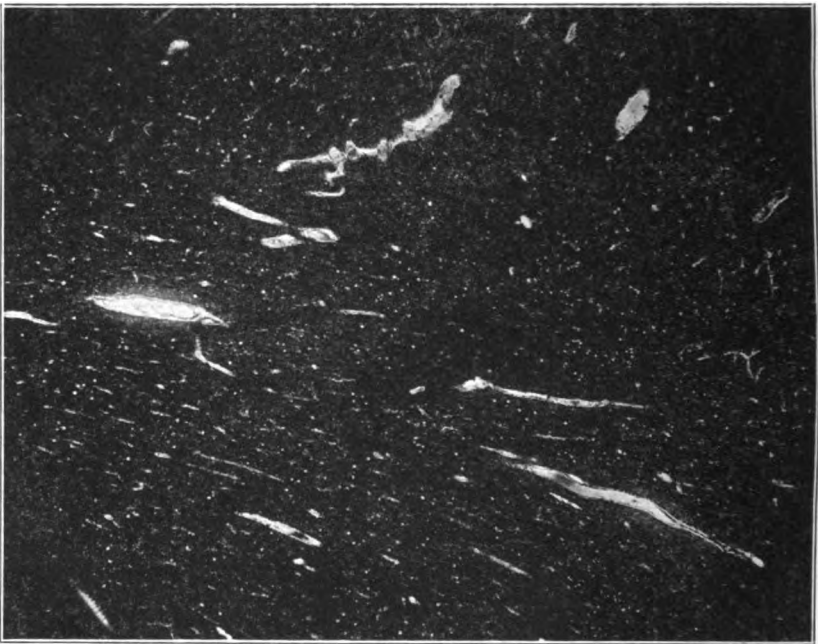


Fig. 9 (Case 2).—Weigert's myelin sheath stain of left motor area showing small foci of degeneration and one larger area in intimate relationship with the neighboring blood vessels.

Her condition gradually grew worse and she was less and less easily aroused. Twelve days after her admission, she died.

Wassermann Reaction: Serum, negative.

Blood Examination: Hemoglobin less than 10 per cent. (Sahli); color index, 1.6; red blood cells, 780,000; white blood cells, 4,700; polymorphonuclear leukocytes, 60 per cent.; small lymphocytes, 32 per cent.; large lymphocytes, 8 per cent. The red blood cells showed marked anisocytosis and poikilocytosis, and some achromia. There was a large number of microcytes and macrocytes. No stipling or nucleated reds were seen.

A later blood examination showed hemoglobin less than 10 per cent. (Sahli); red blood cells, 550,000; white blood cells, 4,500; polymorphonuclear leukocytes, 61 per cent.; small lymphocytes, 34 per cent.; large lymphocytes,

3 per cent.; transitionals, 0; eosinophils, 1 per cent.; mast cells, 1 per cent. Two normoblasts and one megaloblast were seen. The red blood cells showed marked variation in size and shape. There were very many small cells but the majority were macrocytes and the average volume index was increased. There was no achromia and the color index was high. Occasional cells showed marked polychromatophilia and occasional cells showed very marked stippling.

Necropsy Protocol.—The body is that of a well developed, poorly nourished woman, 157 cm. in length. Rigor mortis is not present. No superficial lymph nodes are palpable. The skin is lemon yellow in color. The necropsy was performed thirty-six hours after death. The pupils are equal and regular and 0.4 cm. in diameter.

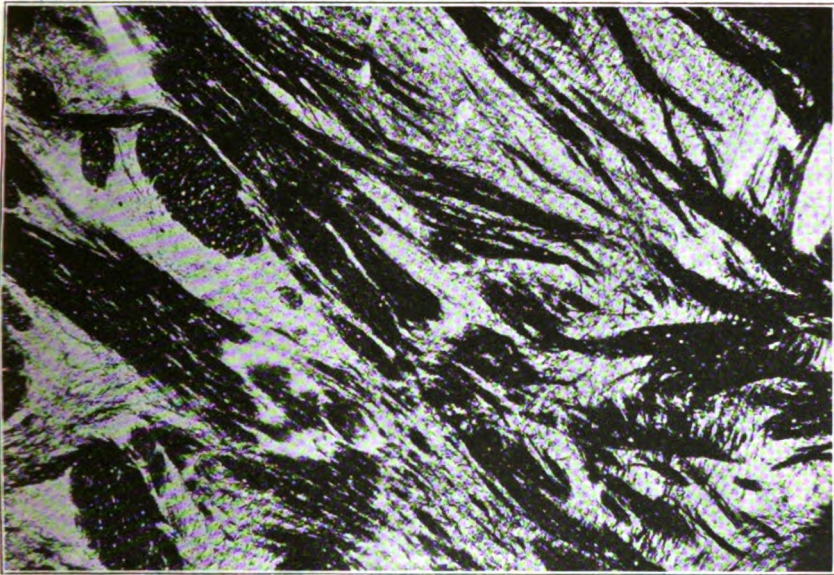


Fig. 10 (Case 2).—A section of the pons stained by Weigert's myelin sheath method. Here again are very clearly shown the miliary foci of Preobrajensky in the medullary substance.

Ventral Section: The abdominal fat is bright yellow; the thoracic and omental fat being bright lemon yellow. The liver is 12 cm. below the ensiform cartilage. The spleen is not encased in adhesions. The appendix is retrocecal and adherent to the cecum. The diaphragm arches to the lower border of the fourth rib on the right and to the lower border of the fifth rib on the left.

Heart: The myocardium of left ventricle shows tiger lily striations with here and there translucent areas. The muscle is soft.

Lungs: These are encased in adhesions. The cut surface is grayish brown in color at the apices. In the lower lobe it is pinkish-yellowish-gray. The connective tissue element is well marked. A frothy, grayish yellow, thick fluid is scraped from the cut surface.

Abdomen: The spleen weighs 185 gm. and is of firm consistency, with a shiny surface. It is purplish brown in color. The capsule is not thickened;

the trabeculae are prominent and the malpighian bodies appear as pin points.

Kidneys: These showed cystic areas on the lateral edge. The capsules stripped easily.

Liver: This weighs 1,560 gm. It is shiny, mottled and of yellowish-brown color. The capsule in general is not thickened. It is of firm consistency.

Pancreas: Pale; otherwise there is nothing of note.

Gastro-Intestinal: The stomach is pale, glassy and atrophic in appearance near the cardiac end.

Head: The skull tables show a symmetrical thickening. There are slight patches of endostosis in the frontal region.

Superior Surface of Brain: There is no apparent atrophy and it is firm to the touch. There are inequalities between the first and second frontals and between the right and left prefrontals.

Base of Brain: The basilar artery is small and no sclerosis in the circle of Willis could be seen. There is a slight thickening of the pia around the third nerves, otherwise the cranial nerves show no abnormalities. The mammillary bodies are flattened. The brain is pale. The fourth ventricle is clear. A pressure ring cerebellum is noted. In the left base the fluid is yellow. This also applies to the cortex. However, neither the right base nor the third ventricle showed this condition. The spinal fluid was mixed with blood. The brain weighed 1,175 gm., which according to Tigges' formula gives a loss in weight of 81 gm.

COLLOIDAL GOLD CHLORID REACTION

	1	2	3	4	5	6	7	8	9	10
Cortex	4	5	5	5	5	3	3	2	1	0
Left base.....	4	4	5	5	5	5	3	2	1	0
Right base.....	1	4	5	3	3	5	3	2	1	0
Third ventricle (blood stained).....	0	0	1	1	2	3	3	2	1	0
Spinal fluid (blood stained).....	0	0	1	4	5	5	3	3	2	
Pericardial fluid.....	0	0	0	1	2	5	5	5	5	5

Histological Examination.—Weigert Section—Left Motor Area.—Small foci of degeneration resembling those described by Preobrajensky are noticed in the subcortical region. These, however, are not numerous. There are also some larger areas of degeneration that are in close relation to the blood vessels.

The cortical perivascular lymph spaces are distended and here and there are associated with small areas of degeneration.

Pons: Here we also see miliary foci of Preobrajensky, but these, unlike those described in Case 1, are more numerous in the peripheral portions of the medullary substance. The myelin is thinned out in many places and in many instances this has gone on to complete degeneration. Three Lichtheim plaques were counted which were in intimate contact with the blood vessels. In addition, two sharply defined areas of degeneration, involving entire tracts, are present. These are also seen in the sections of the pons stained with cresylecht-violet and are more fully described under the latter heading. The gray matter showed practically no involvement.

Medulla: The white matter showed but slight involvement. Discretely scattered between the fibers of the raphe were small foci of degeneration. In the gray matter, however, it was not uncommon to come upon distinct areas of degeneration in the neighborhood of blood vessels. This was especially true of the dorsal portion of the medulla.

Cord: In the left lateral column of the cord, in the cervical region, there are several foci of the Lichtheim type. The blood vessels which are rather

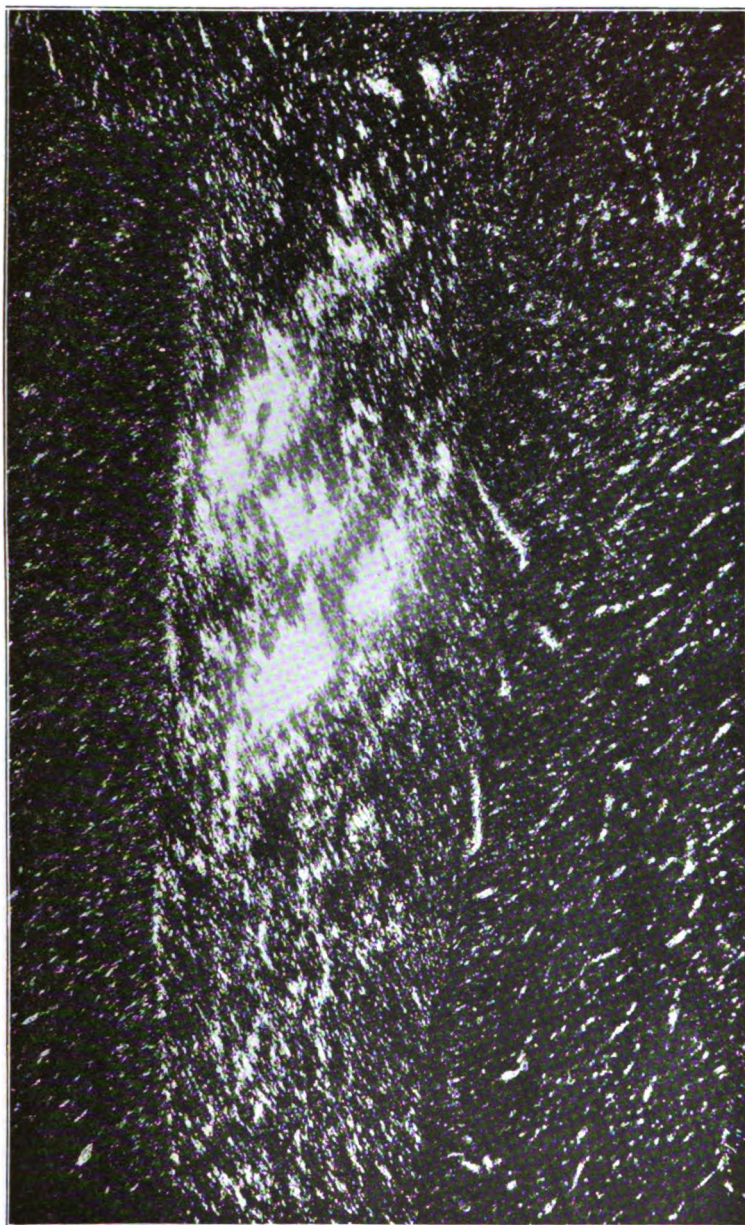


Fig. 11 (Case 2).—Another section of the pons. Note the uneven demyelination of an entire tract. The destruction has progressed very far in the center where large sclerotic areas have been formed. Compare this photograph with Figure 12 which is the same area stained with cresylecht-violet.

numerous have greatly thickened walls. In the lumbar region, there are patchy areas where the myelin is thinned out. The gray matter appears normal.

Cresylecht-Violet Sections—Left Motor Area.—The pyramidal cells, especially those in the outermost layer of the cortex, are shrunken and irregular in outline, the nucleus, in many cases, being crowded to one side. In others it is entirely absent. Satellite cells are numerous. Large stellate glia cells were observed. The blood vessels are numerous and the perivascular lymph spaces are uniformly distended. The subcortical area shows no large areas of degeneration but here and there a few small, clear areas.

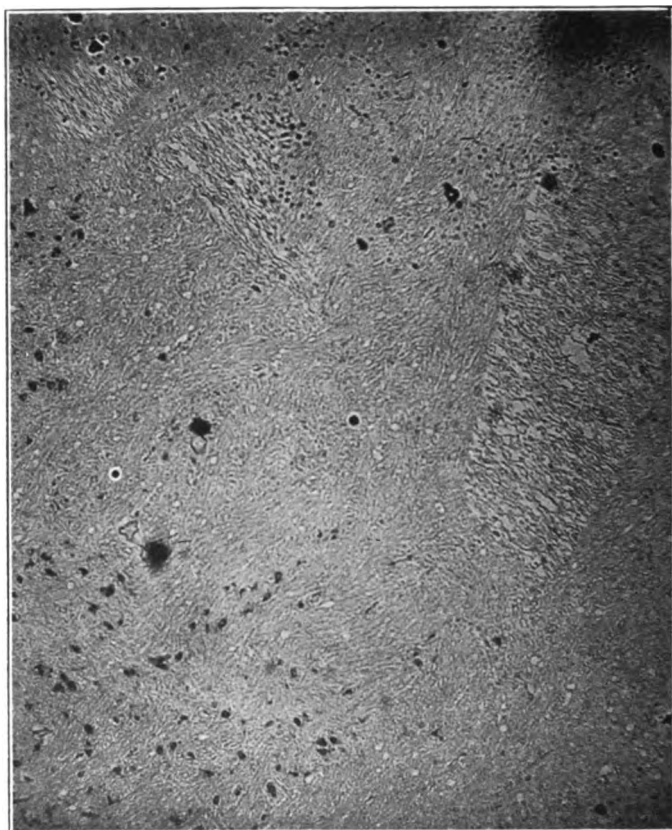


Fig. 12 (Case 2).—Same area of pons as in Figure 11 stained with cresylecht-violet. This also shows the uneven degeneration of the entire tract. To its left, two other foci of degeneration are seen. ($\times 50$)

Pons: Here we see many evidences of a pathologic process. In the medullary substance there are numerous sievelike areas. Here the destruction of the nerve fibers has not been accompanied by any considerable increase in the neuroglia fibers, thereby producing the cribriform appearance. One also notices large sclerotic areas which apparently are composed entirely of neuroglia. These hyaline-like areas involve entire tracts and are definitely circumscribed by fibers of other tracts which evidently have escaped being involved in the destructive process. Furthermore, these sclerotic areas are symmetrically distributed

on either side of the pons. In addition to the areas just described, there are numerous perivascular areas of degeneration. These vary in size and have no definite margins, but shade off insensibly into the surrounding tissue. One of these perivascular areas of degeneration extends into a small collection of nuclear cells which also show evidence of pathological involvement. They are irregular in outline. Many are shrunken and granular and some show a diffuse chromatolysis.

In the larger collections of gray matter, the perivascular spaces are distended and here and there one sees a patchy area of degeneration. These, however, are not numerous. The majority of the nerve cells show more or less changes. The axonal type of degeneration was observed.



Fig. 13 (Case 2).—Cresyl-euchrome-violet sections of the pons. Here there are two sieve-like areas of degeneration surrounding two blood vessels. This shows beautifully the intimate relationship between the blood vessels and the "anemic foci." Evidently in a given area, some of the nerve fibers succumb to the toxic action sooner than others. In this manner, no doubt, the cribriform or sieve-like areas seen above are reproduced. ($\times 50$)

Medulla: In the white matter, there are streaky patches of degeneration. Vacuolization is pronounced. The pyramidal tracts show no changes. Very few of the nerve cells appear normal. The majority are highly granular and show more or less displacement of the nucleus. Clear areas, that appear like rifts in a cloud, are scattered about.

Cord: Many irregular, pale areas, containing necrotic tissue and surrounded by neuroglia, can be seen in the lateral columns. In some cases, the neuroglia



Fig. 14 (Case 2).—Another part of the same section. Here the degeneration, which surrounds the blood vessels, extends into a neighboring collection of nuclear cells. ($\times 50$.)



Fig. 15 (Case 2).—Weigert's myelin sheath stain. Section of the cervical region of the cord showing plaques of degeneration in the lateral columns. The hole in the right lateral column has been artificially produced to indicate the right side of cord. ($\times 10$.)

cells seem to be clumped together. This is also met with but to a much lesser extent, in the posterior columns. The cells of the anterior horns are bizarre in shape and contain yellow pigment, similar to that described in the preceding case.

CASE 3 (Necropsy 18-34).—History.*—J. H., a white boy, aged 6 years and 11½ months, was admitted to the Monson State Hospital, Jan. 15, 1917, with the diagnosis of epilepsy.

Family History.—Father and mother are living and well. The father was 36 and the mother 34 years old at the time of the birth of the patient. The patient is one of seven children. One older brother, who was an epileptic, died. There is no other history of insanity or feeble-mindedness in the family. An older sister of the patient died of pneumonia, aged 2 months. Three older brothers are living and well. The oldest, however, had convulsions when teething at 1 year of age and had four convulsions during the following year. He was then circumcised and he has been free from convulsions ever since. Since the patient was born, one sister has died, aged 6 weeks, cause unknown. One younger brother is well.

Personal History.—The patient had a normal birth and infancy up to 2 years when he had his first convulsion. Teething was normal. He talked at 1 year and walked at 11 months. He has had no exanthemas, except chicken-pox. He went to kindergarten and first grade, passed and got along as well as the other children.

Mental Condition.—Although the mother states that the patient went to kindergarten and passed the first year in graded school, he is defective in school knowledge of the first grade. He can count up to 100 but can write only the initials of his name. He can read and write no words at all. He reacts normally to the emotions and shows no other abnormal reactions except an apparent memory defect for daily occurrences. He behaves rather strangely at times, apparently not realizing just where he is going or what he is doing when out with other boys. He starts off aimlessly and apparently needs supervision. Binet tests, however, show his mental age to be 6.1 years. According to the Yerkes-Bridges scale he is 7.1 years mentally. His first seizure occurred at 2 years of age. According to the mother's statements, the patient was in a baby carriage which was being wheeled by a young girl when the carriage suddenly tipped over. The child was apparently unhurt. Eight hours later, however, he had a convulsion which the mother attributes to the fall. There is no history of unconsciousness or apparent injury at the time of the accident. Since that time, however, the patient has had seizures, both of grand mal and petit mal type. These, furthermore, have been increasing in frequency of late and at present occur almost daily.

Physical Examination.—The patient shows no marked asymmetry. Head measurements: circumference, 20 inches; glabella toinion, 11 inches; transverse, 10 inches. The neurological examination showed nothing abnormal. The blood Wassermann reaction was negative.

Course.—The patient remained at the hospital until June 27, 1916, and during that time had on an average ten convulsions per month. He gained in weight and height, but had made little or no progress in school. He had an attack of status epilepticus of twenty-six grand mal seizures. He was readmitted to the hospital on Oct. 20, 1917, without any apparent change in his physical condition. He had had an attack of status epilepticus while home on

* This case will be reported more fully in a later paper.

a visit. A second Wassermann test was reported negative. After readmission he had less seizures, averaging only two or three each month, but he exhibited a marked change in his mentality. He attended school but without making any progress. At times he would play and associate with other children, sing songs which he had committed to memory and appear quite normal. At other times, lasting for periods which varied from a few hours to several days, he would be very seclusive; hide himself about the ward, in closets, under beds; would not associate with the other children, but would be found sitting alone quietly and if asked the reason for so doing would reply, "Other boys are rough and intend to kill me." Very often he would volunteer the information that he was going to kill himself but would not specify in what manner.

There was no marked change physically or mentally until April 16, 1918, when the patient was noticed to be suddenly acutely ill. He vomited several times, complained of headache, and there was a slight rise in temperature.

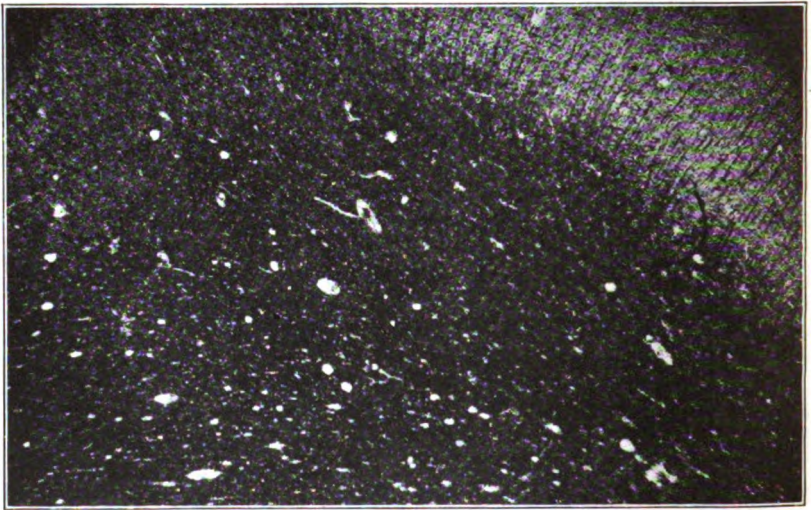


Fig. 16 (Case 3).—Section of the left motor area showing numerous foci of degeneration which vary greatly in size. Weigert's myelin sheath stain.

By the 21st the condition had somewhat improved. There was no nausea or vomiting and the patient took liquid nourishment fairly well. The skin of the whole body had a marked greenish, lemon yellow tint. The mucous membranes showed marked pallor. A blood examination showed hemoglobin, 70 per cent.; 70,000 leukocytes and 2,000,000 red blood cells. The color index was high, namely, 1.75. A differential count gave polymorphonuclear leukocytes, 57 per cent.; small lymphocytes, 20 per cent.; large lymphocytes, 21 per cent.; transitionals, 1 per cent.; basophils, 1 per cent. A differential stain showed granular degeneration of the red cells, presence of nucleated reds, and also some megaloblasts.

In the night the patient was very restless and exhibited a marked psychosis. With his finger nails he tore a gash in the perineum, anterior to and extending into the rectum. He also scratched his face and hands with his finger nails.

On the 24th he was more quiet but still threatened to destroy himself and had to be restrained. The skin still retained the same peculiar hue and there

were no different physical signs. Toward evening the temperature rose to 103 F., and early the next morning the patient died.

Cause of Death: Pernicious anemia.

Necropsy Protocol.—Necropsy was performed ten hours after death. *External Examination:*—The body is that of a slenderly built, poorly nourished, white male child, 9 years of age. The skin has a bluish gray appearance over the abdomen and lower chest, with yellowish cast of all muscle depressions over the chest and neck. There is a faint yellowish tinge to the sclerae. The sub-maxillary glands are prominent. The lymph nodes posterior to the sternocleidomastoid as well as the axillary and inguinal glands are palpable. The pupils are unequal, the right being 2 mm. and the left 4 mm. in diameter. The teeth are also unequal in size. Rigor mortis is present. The body length is 127 cm.

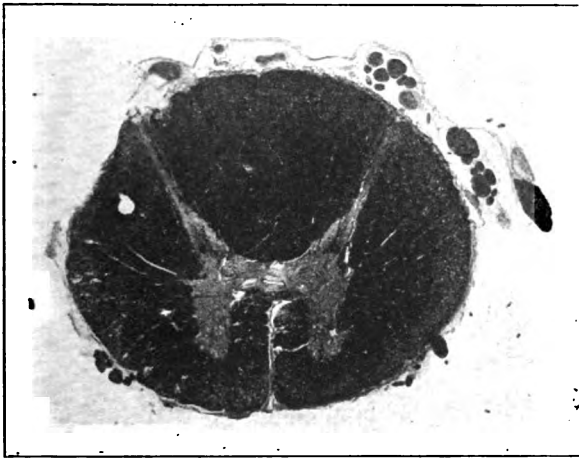


Fig. 17 (Case 3).—Section of the dorsal region of the spinal cord. The right lateral column is principally involved. Some degeneration changes have also occurred in the anterior columns. ($\times 10$.)

Ventral Section: The fat over the abdomen is of a pale lemon color and moist. The muscles are red. The lower border of the liver is 5 cm. below the ensiform cartilage. It is brown in color. The spleen is large and free. The appendix is also free and measures 8 cm. in length. The mesenteric lymph nodes are enlarged and some are calcareous. The diaphragm arches to the third interspace on the right and to the fourth interspace on the left.

Heart: The heart muscle is firm, grayish pink in color and shows no white areas or fat.

Lungs: Both lungs showed slight congestion in the posterior portions of the lower lobes.

Organs of the Neck: The thymus is present. The tissue is stringy, but very little of the gland tissue is left.

Abdomen: The spleen shows three fetal lobulations. The pulp is firm and the malpighian bodies numerous. The kidneys show nothing abnormal. The liver weighs 1,240 gm., and a cut section looks greenish yellow. It is homogeneous, there being very scant outlining of the lobules.

Gastro-Intestinal: There were depressions in the gastric mucosae suggesting beginning ulcerations.

Head: The skull is pale in color and shows irregular thinning over the vertex and frontal region.

Brain: The dura is adherent to the calvarium. The cortex shines through a thin and delicate pia mater which is gray. The cranial nerves appear smaller than usual. The frontal lobes appear out of proportion to the parietal. The temporal tips are unequal in pattern, the left being more complex than the right. The hemispheres of the cerebellum are also slightly unequal, the left being the larger. The brain substance is firm, especially the right frontal region. The weight of the brain is 1,280 gm., which represents a gain of 264 gm., according to Tigges' formula.

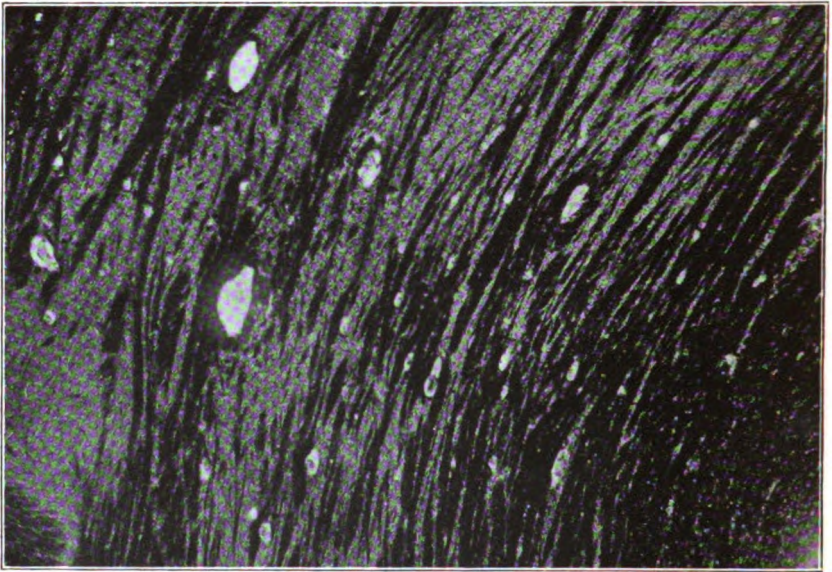


Fig. 18 (Case 3).—Just as in the two preceding cases, this section of the pons also shows the miliary foci of Preobrazhensky. Weigert's myelin sheath stain.

Cord: The cord shows "china white" softening in the posterior columns, especially in the lumbar region.

Histological Examination.—*Weigert Sections—Left Motor Area:* In the subcortical areas, the perivascular lymph spaces are distended and clear. Many small discrete foci of degeneration and four larger sclerotic plaques were noted. The cortex itself does not present anything unusual.

Pons: This section, like the two preceding sections of the pons, also contains the miliary foci of Preobrazhensky. However, they were not nearly as numerous nor as universally distributed, being limited to a small portion of the medullary substance. The blood vessels in the nuclear masses are thickened and filled with red blood cells. The perivascular spaces are markedly distended, and some also contain red blood corpuscles.

Medulla: Here there is only slight evidence of pathological involvement. The lesions found are similar to those described above.

Cord: (Cervical region).—In the lateral column, near the anterior horn, there is a well-defined focus of degeneration of the Lichtheim type. The lateral cerebellar tracts show patchy myelin sheath degeneration, with here and there evidence of vacuolization. (*Dorsal region*).—Here the lesions are even more marked. The anterolateral columns show great involvement. Many foci of degeneration, including a Lichtheim focus, are seen. The blood vessels do not show any abnormalities. (*Lumbar region*).—This section shows nothing of note.

Cresylecht-Violet Sections—Left Motor Area.—The principal abnormality noted was on the part of the pyramidal cells. A large number of these cells were shrunken and the contour of their nuclei was not clear cut. Some were highly granular and some were surrounded by satellite cells. Here and there in the subcortical area there were small vacuoles.

Pons: Here the changes in the nerve cells are similar to those just described, only much more accentuated. The axonal type of degeneration was observed. The larger pyramidal cells did not appear to be as greatly involved as the smaller.

Medulla: Here one notices large sclerotic areas which are apparently closely identified with the blood vessels. Small, clear, hyaline-like areas are fairly numerous. The nerve cells show but insignificant changes.

Cord: (Cervical region).—The blood vessels are slightly thickened and the perivascular spaces are distended and filled with hyaline-like material. (*Dorsal region*).—Here we meet with ballooning of the nerve fibers with the formation of clear sievelike spaces. There does not appear to be an increase in the neuroglia. Many of these irregular areas are in close proximity and even surround the blood vessels. The cells in the anterior horns show a high degree of chromatolysis. Some of the cells appear to be entirely disintegrated. (*Lumbar region*).—In this region there is very little of a pathological nature to be seen.

CASE 4.—(Necropsy 16-52.—*History.*—J. F., aged 53, was committed to the Northampton State Hospital for the Insane, May 10, 1915. The medical certificate reads as follows: "Patient is recovering from an acute exacerbation of a chronic nephritis. Patient has a cross on his arm, which he hopes will prove certain things if he can see the priest. Wishes to see the selectmen about certain property and wishes to tell them that while there is life there is hope; that the sky is blue and other such things. Wishes to see a lawyer about disposing of his property. Is an inmate of almshouse and has none."

General Appearance.—The patient is a well-nourished, simple looking man, over 50 years of age, very anemic in appearance. During the examination he was quiet, but listless and indifferent. He did not volunteer conversation, replying correctly, however, when directly questioned. There was no stereotypy, autonomy, blocking or flight of ideas, or other abnormalities noticeable, except possibly a slight retardation. This may be his normal reaction, as he does not appear overbright.

Family History.—His father is said to have been insane. One brother is in Baldwinville (hospital for chronic infantile neurological cases), and another brother was in the Northampton State Hospital, the diagnosis in his case being manic depressive insanity.

Personal History.—The patient was born and brought up in Ware, Mass., where he spent all of his life except for short occasional absences. He can only read and write. He was never employed except as a common laborer. He

worked, however, only intermittently, and for the greater part of his life was looked after by his sister. For the past several years he has been in poor physical health. He cannot state anything exact concerning this, but says that his appetite has been poor and that he believes he has kidney trouble. At one time he is said to have been very intemperate, but denies drinking to any extent for several years prior to his admission to the hospital. In 1910, he was committed to the Howard (R. I.) State Hospital. The diagnosis at that time was delirium tremens. Three years later he was again admitted to the Howard State Hospital, where a provisional diagnosis of chronic alcoholism was made. The mental examination at that time, showed depression and



Fig. 19 (Case 4).—Numerous areas of degeneration of varying sizes are shown in this picture of the cortex. In several, the close relationship to the blood vessels is seen. Weigert's myelin sheath stain.

defective knowledge of current events. The patient denied hallucinations, but it was stated that a short time before coming to the hospital he heard noises on waking from sleep. He was discharged in 1914 and returned to his home from where he was sent to the Ware Almshouse. From there he was committed to the Northampton State Hospital.

Physical Examination.—A summary of the physical examination reads as follows: "Fairly well nourished male with rather poor muscular development and of decidedly anemic appearance. The mucous membranes are very pale. There is a complaint of dyspnea on exertion and of discomfort after eating. Appears very much like pernicious anemia."

Neurological Examination.—There are no unusual phenomena observed. The patient is quiet on examination. All motions are slow and deliberate. There is no tremor or flush.

Muscle Power: This is poor.

Coordination: There is a slight swaying in the Romberg position, but he is able to stand without support. The gait is normal. Coordination tests were fairly well, although slowly performed.

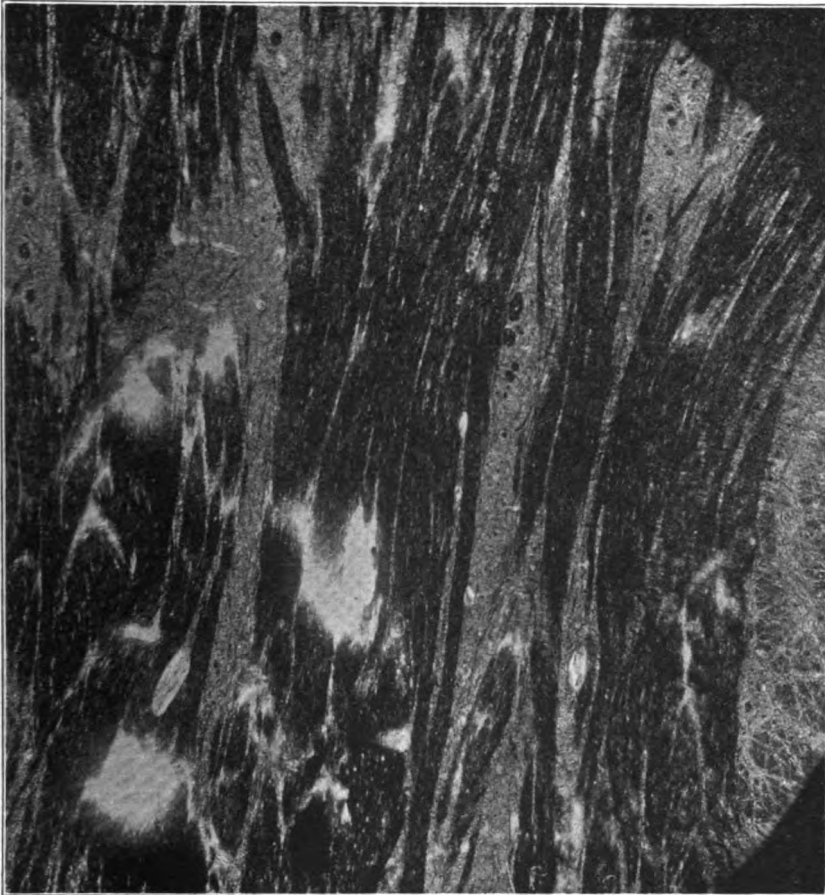


Fig. 20 (Case 4).—Weigert section of the pons. In this photograph, the Lichtheim type of focus predominates. A few of the military foci of Preobrazhensky are also present. Note the distention of the perivascular spaces.

Sensation: The feet and hands are slightly cooler than the other portions of the body. Stereognostic sense is normal.

Cranial Nerves: The pupils are normal in size and equal and react to light and accommodation. All the other cranial nerves are normal.

Reflexes: The superficial reflexes are entirely absent. The knee jerks are absent. The other tendon reflexes are very faint. There are no abnormal reflexes.

Mental Examination.—Orientation: Good in all spheres. Memory: Remote and recent memory rather poor. He remembers things in general ways but cannot give details nor dates, and appears somewhat demented.

Education: He is very poorly educated. He can read and write, but has little general knowledge, and no knowledge at all of current events.

Delusions: On admission, he talked of vague unsystematized, indefinite delusions of owning considerable property which was due him from his father's estate. Mild delusions of persecution against his sister were also present, he believing that she had deprived him of his share of the estate. He made other vague statements, but his whole delusional formation was very loosely connected. He talked only when directly questioned and gave the impression of being considerably deteriorated.



Fig. 21 (Case 4).—Section of the cervical region of the spinal cord. The posterior and postero-lateral columns are involved. A narrow strip on either side of the posterior fissure has escaped involvement in the pathologic process. Weigert's myelin sheath stain. ($\times 10$.)

Hallucinations: No definite hallucinations could be elicited. The abstract from the hospital at Howard, R. I., states that before his admission there he was hearing noises on waking from sleep. This may possibly have been a mild hallucinosis following drink.

Emotional Tone: He takes very little interest in anything about him. He shows neither exhilaration nor depression, but always appears indifferent.

Demeanor: Since his admission he has been quiet and fairly cheerful. He never volunteers conversation with any one but answers when directly questioned.

Attention: This is fair.

Abstract of Ward Notes.—On admission, he was correctly oriented. He remembered that his brother had once been a patient at the hospital. The

patient complained of not feeling well physically. He believed he had kidney trouble. He talked vaguely of his delusions and denied that he had been intemperate in recent years. He was quiet and conducted himself well. At first he helped a little in the ward when requested, but made no attempt to associate with any one, although he could not be considered surly. When

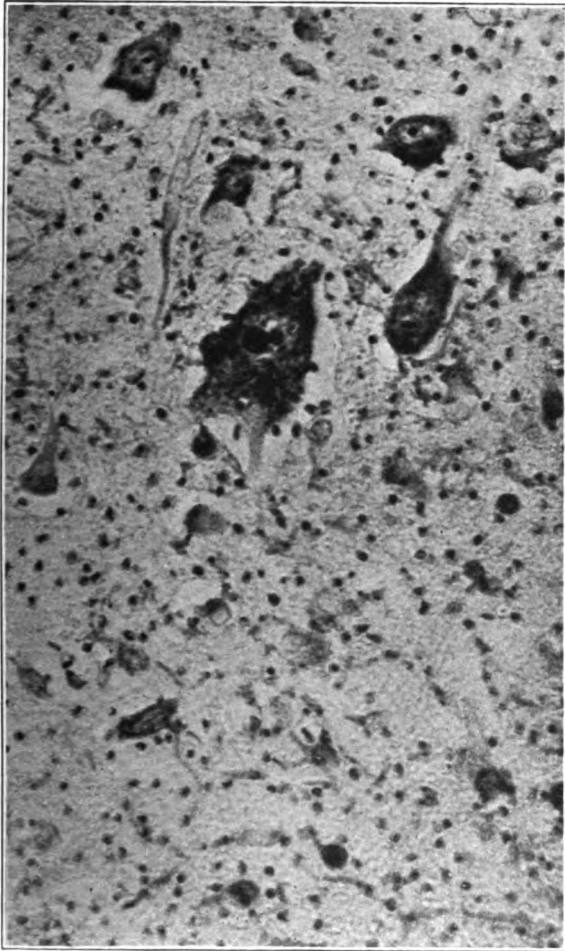


Fig. 22 (Case 4).—This is a section of the cortex stained with cresyl-euchrome-violet. A large pyramidal cell (neuronophage) can be seen which is evidently greatly swollen and undergoing degeneration. It contains two glia cells, one being within the cell body and the other within the nucleus in close contact with the nucleolus. ($\times 100$.)

addressed, he would reply but would not continue the conversation. He took no interest in games, in reading or in the discussion of his fellow patients. Whether this listlessness was due to deterioration or to his poor physical condition was difficult to determine.

In June, 1915, he began to complain of epigastric pain and of a feeling of heaviness following the taking of food. In September, a slight dyspnea was noticed on exertion and the epigastric distress had become so marked that he could only take malted milk, vomiting every other food. The skin showed the peculiar brown tinge peculiar to pernicious anemia. A single blood count showed 3,260,000 red blood cells and a hemoglobin of 60 per cent. Numerous poikilocytes, microcytes, and macrocytes were seen, but no nucleated reds were seen at this time. There was puffiness about the eyes. Some days he complained of weakness in the legs.

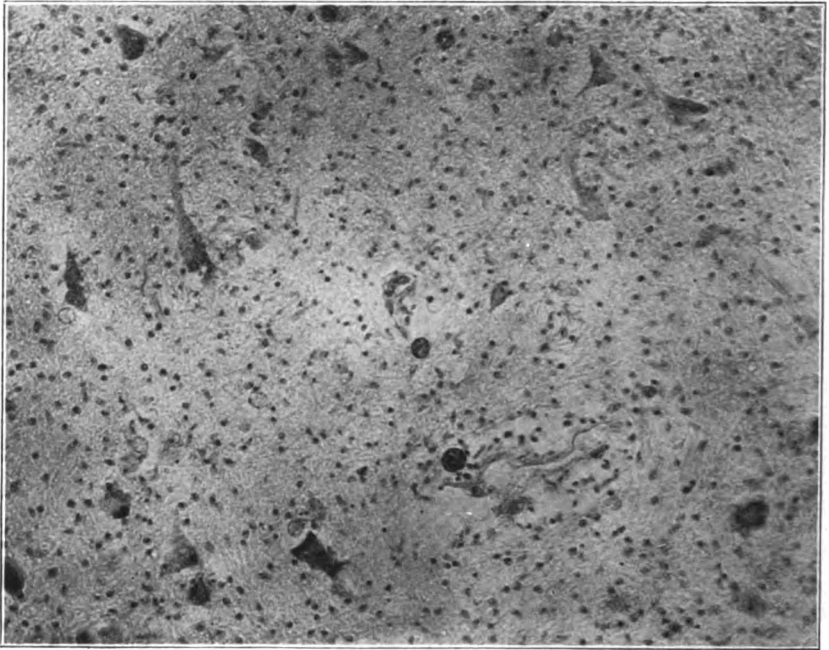


Fig. 23 (Case 4).—Another view of the cortical motor area showing disintegration of the pyramidal cells, and distention of the pericellular spaces. Some of these cells are pale, shrunken, irregular in outline, stain poorly and unevenly, and show eccentricity of the nucleus. The severe neuroglial reaction is evident. In the lower right hand corner of the field, note the large sclerotic area surrounding a blood vessel. Cresylecht-violet stain. ($\times 50$.)

In October, physical failure became more marked. The dyspnea was so intense that care in bed became necessary. Puffiness of the eyes, hands, and feet was very noticeable. Mentally he was very dull, apparently comprehending but little. In November, 1915, a note in the history was made that he seemed slightly improved, which temporary improvement continued until April, 1916. In January, 1916, typical findings of pernicious anemia were present in the blood and especially the nucleated red blood cells. Early in April he failed rapidly and died on April 15, without showing any new development of his disease.

Necropsy.—Protocol (16-52).—Necropsy was performed two hours after death. The body is that of a white male, 168 cm. in length. The skin is pale

lemon yellow, especially over cheeks, forehead, and arms and legs. There is considerable pigmentation over the back, chest, face and lower abdomen. Rigor mortis is present in jaws and legs. The lymph nodes are not palpable. The pupils are equal and measure 0.4 cm. in diameter. The eyeballs are slightly softened. The teeth show Riggs' disease.

Ventral Section: The fat over the abdomen is pale yellow and moist. The muscles are red and mixed with apparent fatty streaks. The lower border of the liver is 1 cm. below the ensiform cartilage. There is a slight amount of free fluid in the flanks. The appendix is 8 cm. in length. The tissue in the pelvis appears bloodless. The spleen is surrounded by adhesions. The diaphragm arches to the third rib.

Thorax: The sternal marrow is richly red and somewhat fluid. There is a free fluid in the left chest and the pericardial sac is thickened.

Heart: The epicardial fat is abundant and there are milk spots on the posterior surface. The heart muscle shows white mottling and also minute hemorrhages in the right auricle. It fragments easily.

Lungs: They show very little of note.

Abdomen: The spleen is grayish red and its capsule is slightly withered. A section shows the pulp to be red. It measures 10 by 5 by 2 cm.

Adrenals: These are small and softened.

Kidneys: There is an excessive amount of fat around the kidneys which are yellowish brown in color. The pyramids are white and poorly differentiated from the surrounding tissue. Two cysts containing fluid are seen.

Liver: There are slight irregularities over the surface of the liver which is yellowish red in color. A section shows packing together of the lobules. It measures 23 by 16 by 7 cm.

Pancreas: This organ is dotted with hemorrhages.

Gastro-Intestinal: The stomach is large and contains some fluid. The mucous membrane of the stomach is shiny.

Brain: The dura shows signs of absorption in the frontal region and of thickening along the longitudinal sinus. A slight amount of yellow fluid escapes on section of the dura. Points of hemorrhages are seen in the pia mater which shows some thickening along the vessels. It is held up from the cortex by fluid in the motor regions. The brain appears yellowish white. The right lobe sags, being apparently slightly shorter than the left. The pattern of convolutions is more nearly circular over the right lobe than over the left lobe. The brain has a resilient feeling. From the basal aspect, it is noticed that the pia is thickened over the pons, left third nerve, and optic chiasm. The left temporal tip is softer than the right. The left cerebral artery is larger than the right. The fourth ventricle is clear.

COLLOIDAL GOLD REACTION

	1	2	3	4	5	6	7	8	9	10
Right base.....	0	0	0	1	2	1	0	0	0	0
Left base.....	0	0	0	1	1	1	0	0	0	0
Third ventricle *.....	0	0	0	0	0	0	0	0	0	0
Spinal fluid.....	0	0	0	0	0	0	0	0	0	0
Pericardial fluid.....	0	0	0	0	0	0	0	0	0	0

* Slightly bloody.

Histological Examination.—Weigert Section—Left Motor Area.—The medullary substance shows little of note. There are a few areas where the myelin has been completely destroyed and also a few places where there is a thinning out of the myelin.

Paralleling the edge of the cortex, there is a narrow strip of tissue which stains less heavily than the surrounding tissue. Under high power, this area is shown to contain small irregular shaped spaces which for the most part are structureless. A few are crossed by undegenerated or only partially degenerated nerve fibers. In this area, furthermore, the capillaries are very numerous and the perivascular spaces are distended.

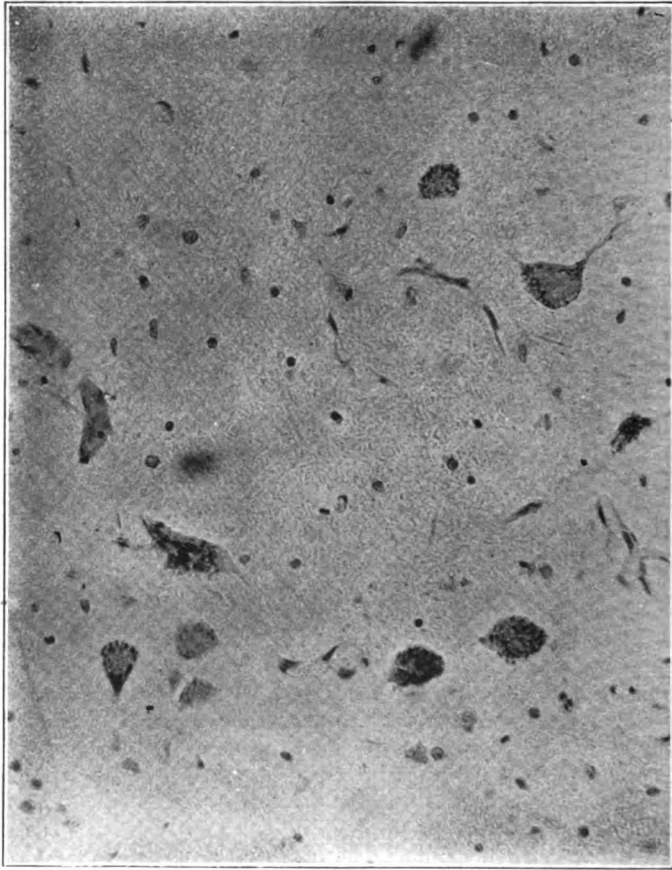


Fig. 24 (Case 1).—Section of cervical region of spinal cord stained with cresylecht-violet showing some anterior horn cells containing yellow pigment. This pigment is diffuse, slightly granular, and occupies the central portion of the cell body, the protoplasmic substance being pushed to the extreme periphery. The two cells in the lower left hand corner of the field are practically nothing but a mass of pigment. ($\times 100$.)

Pons: Here we see large foci of destroyed tissue in intimate relation with the blood vessels. It is almost possible to trace the entire process of their formation as they are present in all stages of development. One notices first a slight thinning out of the myelin sheaths in the immediate neighborhood of a blood vessel. The process continues and this thinned out area becomes

sievelike, due to the lack of uniformity in the destruction of the myelin sheaths. In the meshes of some of these cribriform areas, red blood cells are found. In the more advanced places, there has been an increase in the neuroglia with resultant formation of large plaques of sclerotic tissue. In one part of the field, the miliary foci described by Preobrajensky were also observed.

Medulla: Small ragged foci of destroyed tissue, irregularly distributed are seen. The perivascular spaces of the blood vessels in the olivary nuclei are markedly distended.

Cord: (Cervical region).—In this region, there are numerous small foci where the myelin sheaths have been destroyed with subsequent vacuolization. This destructive process has occurred principally in the columns of Burdach. The columns of Goll are but slightly affected. The direct pyramidal tracts show more or less degenerative changes also. **(Dorsal region).**—This region appears practically normal. In the gray matter of the anterior horns one can see several small hemorrhagic areas. This is also present in the lumbar region which otherwise shows nothing of note.

Cresylecht-Violet Sections:—Left Motor Area.—The cortical region shows extensive pathologic involvement. There is a tremendous overgrowth of neuroglia both of the fibers and cells. The latter are encroaching on the pyramidal cells. The glia cells are of two kinds: (1) a small and apparently homogeneous cell, and (2) a large cell with a granular nucleus which in many ways resembles a small lymphocyte. In several instances, these cells both large and small are seen to be incorporated in the body of the pyramidal cells and in one instance one of these larger cells is seen to be incorporated within the nucleus of the nerve cell. The nerve cells, with but few exceptions, show marked degenerative changes. They are shrunken, irregular in outline, and the protoplasm is not uniform in structure. The nuclei show chromatolysis and their outline is hazy. Their position varies, being either to one side or at the end of the cell. Some of the cells instead of being shrunken are tremendously swollen. In some cases, the nerve cell seems to have disappeared entirely or fallen out, the pericellular spaces being occupied by glia cells.

The blood vessels also show a severe reaction. The perivascular spaces are distended. The capillaries show a tremendous increase. One sees them in all stages of development, from the small, budding, rod-like projection to the thin-walled vessel with an almost imperceptible lumen.

All these pathologic changes gradually decrease in intensity as we approach the subcortical area. In the latter structure, there are many clear areas in which no nerve fibers are seen. These areas are irregular in size and shape. In the larger ones, glia fibers have replaced the nerve fibers, giving the area a hyaline-like appearance. This is brought out very distinctly with the eosin-methylene blue stain.

Right Peduncle: There is a uniform distention of the perivascular spaces, many of which are contiguous to foci of degeneration. Some of these areas are cribriform or sievelike in appearance. The pyramidal cells show evidence of involvement. Some are completely disintegrated, some have lost their nuclei and are highly granular, and others are pigmented. Satellitosis is marked.

Pons: Small, clear, fairly regular areas corresponding, in all probability, to the miliary foci of Preobrajensky that were seen in the Weigert Section, are present in the raphe. The nerve cells do not show as great an involvement as those in the preceding sections. Only a comparative few are entirely disintegrated and none show pigmentation. The perivascular spaces are distended. There are also patches of neuroglia overgrowth.

Medulla: The nerve cells in the different collections of gray matter show uniform degeneration of varying intensity. The pericellular spaces are enlarged. Pigmentation occurs frequently. Some of the blood vessels show a thickening of the intima and a small number are surrounded by small areas of degeneration.

Cord: (Cervical region).—There is a sharply defined sclerotic area in the posterior column which is identical with that seen in the Weigert Section. This area is composed chiefly of massive wavy bundles of neuroglia fibers with which are intermingled an occasional undegenerated or only partially degenerated nerve fibril. The nerve cells in the posterior horns appear shrunk in size and the nuclei do not stain well. The anterior horn cells show some pigmentation. This is especially true of those in the lumbar region. The character and location of this pigment are different from that of the pigment normally present in the cells of people 50 years or older. In this case, the pigment is granular, and diffusely and evenly distributed over the central portion of the cell.

DISCUSSION

A brief résumé, contrasting the clinical with the pathologic findings reveals a fairly uniform and definite relationship. In Case 1, we can assume from the history that the condition had probably existed for about three years although a blood examination was made only five days before death. On the clinical side we find a typical blood picture of pernicious anemia; absence of all the superficial reflexes with the exception of the left inguinal reflex; absence of both patellar reflexes and sensory disturbances in the form of numbness of the feet. Mentally, the patient had visual hallucinations, paranoid ideas and mild delusions of persecution. The necropsy report showed the characteristic changes on the part of the heart and stomach. Contrasted to this clinical picture there are definite pathologic findings. In the motor area we find characteristic vascular changes, pyramidal cell changes, satellitosis, vacuolization and the presence of the Lichtheim foci of degeneration. In the pons we have in addition to the above changes the miliary foci of Preobrajensky. In the spinal cord, the posterior column is practically entirely destroyed especially in the cervical region. Neural and vascular changes are also present. Pigmentation of the cells is pronounced and the neuroglia changes marked.

In Case 2 there is a definite blood picture of pernicious anemia. From the history we can safely infer that the condition had existed for many years. Clinically, there are neurological disturbances in the form of hyperesthesia of the lower extremities and an unsteady gait and mental symptoms similar to Case 1, namely: visual hallucinations, delusions of persecution, and paranoid ideas. The necropsy examination revealed a pale, glassy atrophic stomach and a heart with tiger-lily striations. Histologically, the cortex showed areas of degeneration

with vacuole formation, vascular changes, pyramidal cell changes, satellitosis and a marked increase in the neuroglial elements. The pathology of the pons was practically the same as that of the cortex, but again with the addition of the miliary foci of Preobrajensky. The spinal cord presented practically the same pathologic picture as the first case.

In Case 3 the clinical picture is somewhat different. In the first place the patient was very young, being about 9 years old at the time of his death; in the second place he was a decided epileptic, and in the third place the onset of the pernicious anemia was acute. Hence, clinically, we have comparatively few findings. There were no neurological changes, but mentally he showed delusions of persecution and paranoid ideas. During the height of the fatal attack, he showed a marked psychosis. On necropsy, there were slight changes in the stomach, resembling beginning minute ulcerations. The heart was negative. The brain showed a gain in weight, according to Tigges' formula, of 264 gm. However, Tigges' formula is not strictly applicable to children. Pathologically, in the cortex, the changes were similar to those in the first case except that they were less marked. No neuroglial changes were seen which was to be expected, considering the short duration of the disease. On the whole, the changes in this case were the least marked. Here again, however, the miliary foci of Preobrajensky were found in the pons.

In Case 4 the process evidently had gone on for several years. The blood picture was typical. There were marked neurological disturbances, all the superficial reflexes and both knee jerks being absent. There was also a distinct weakness of the legs. Mentally, there were paranoid ideas and delusions of persecution and other vague unsystematized delusions. No definite hallucinations were elicited. On necropsy the heart showed milk spots, white mottling, and minute hemorrhages. The mucous membrane of the stomach was shiny. The histopathologic changes were similar to those in Case 2. No Lichtheim focus was seen in the cortex. There was a marked increase in the neuroglial elements. Neuronophagic action of the cells was marked. The pons showed the miliary foci of Preobrajensky, the Lichteim foci, vascular changes and slight nerve cell changes. In the spinal cord, the columns of Burdach showed the greatest involvement. The nerve cell changes were slight. Pigmentation was present. Also vascular changes. There was a considerable increase in the neuroglia.

The following tables show these results in tabular form:

TABLE 1.—SUMMARY OF CLINICAL FINDINGS IN THE CASES REPORTED

	Case 1	Case 2	Case 3	Case 4
1. Duration:				
(a) From symptomatic standpoint.....	3 years	Many years	Acute	Several
(b) Laboratory standpoint.....	5 days	1 year	Acute	1 year
2. Blood picture.....	+	+	+	+
3. Neurologic Findings:				
(a) Pupils.....	Normal	Sluggish	Normal	Normal
(b) Superficial reflexes.....	All absent except left inguinal	Not stated	Normal	Absent
(c) Knee jerks.....	Absent	Sluggish	Normal	Absent
(d) Abnormal reflexes.....	None	None	None	None
(e) Sensory disturbances.....	Numbness of feet	Hyperesthesia of lower extremities; unsteady gait	None	Weakness of legs
4. Mental Findings:				
(a) Hallucinations (visual).....	+	+	None	Doubtful
(b) Delusions of persecution.....	+	+	+	+
(c) Paranoid ideas.....	+	+	+	+
5. Necropsy Findings:				
(a) Skin.....	Lemon yellow	Lemon yellow	Yellowish	Lemon yellow
(b) Fat.....	Lemon yellow	Lemon yellow	Lemon color	Pale yellow
(c) Marrow: (a) Sternum.....	Yel. pink	Not given	Not given	Richly red
(b) Femur.....	Raspberry red			
(d) Heart.....	Enlarged white striation	Tiger lily striation; no enlargement	Normal	No enlargement, milk spots, white mottling; hemorrhages
(e) Spleen.....	165 grams	185 grams	160 grams	13.5 × 10 × 1.5 cm.
(f) Liver.....	1,800 grams	1,560 grams	1,240 grams	28 × 16 × 7 cm.
(g) Stomach.....	Thick walls; glossy; no rugae	Pale, glossy, atrophic	Ulcerations?	Mucous membrane shiny
(h) Brain.....	Loss 308 grams	Loss 81 grams	Gain 264 grams	Not given
(i) Cord.....	Minute specks of translucency in posterior columns	Not stated	China white softening in posterior columns	Not stated

TABLE 2.—SUMMARY OF HISTOLOGIC FINDINGS IN THE SERIES OF CASES REPORTED

	Case 1	Case 2	Case 3	Case 4
1. Motor Area:				
(a) Vascular changes.....	+	+	+	+
(b) Lichtheim foci.....	+	Not definite	None	None
(c) Vacuoles.....	+	+	+	+
(d) Pyramidal cell changes.....	+	+	+	+
(e) Satellitosis.....	+	+	+	+
(f) Neuroglia changes.....	Slight	Marked	None	Marked
2. Pons:				
(a) Military foci of Preobrajensky.....	+	+	+	+
(b) Lichtheim plaques.....	+	+	None	+
(c) Nerve cell changes.....	Not marked	+	+	Slight
(d) Vascular changes.....	+	+	+	+
3. Spinal Cord:				
(a) Posterior column degeneration.....	++	Very slight	+	+
(b) Lateral column degeneration.....	+	+	+	Slight
(c) Vascular changes.....	+	+	+	+
(d) Nerve cell changes.....	+	+	+	Slight
(e) Pigmentation.....	+	+	None	+
(f) Neuroglia changes.....	Marked	+	None	+

From the above tables, it is evident that the neuropathology of the brain in pernicious anemia is larger and more fruitful than that of the cord. It is true, of course, that all these cases showed very definite mental symptoms and hence one would naturally expect to find cortical

changes. However, in the case of J. H. (Case 3) who had a definite psychosis with no neurological disturbances, the cord changes were by far more marked and had progressed much farther than the cortical changes. From this one might argue that in pernicious anemia, the first degenerative changes occur in the cord; then the process gradually extends and involves the brain.

In the third case, there was also very little involvement of the neuroglia. This, as well as all the other points in which this case differed from the others, can be readily explained on the ground that this case had an acute onset and ran a very rapid course.

The significance of the presence of the miliary foci of Preobrajensky in every section of the pons is open to speculation. Are these lesions specific for pernicious anemia or is their uniform presence in the pons merely a coincidence? Obviously, one should not generalize from the findings of only four cases. However, to look on the regularity of their presence as merely a coincidence seems to me to be unjustifiable. At any rate, it is a point worthy of note and of further investigation.

CONCLUSIONS

1. There appears to be a fairly definite and constant relationship between the clinical symptoms and the pathologic changes.

2. The psychoses can be classified with the symptomatic psychoses of a toxic-organic nature. The whole delusional formation is vague, unsystematized and loosely connected.*

3. The brain changes are even more marked than the cord changes provided the disease has existed for a considerable length of time. This, in my opinion is due to the fact that in addition to the toxic action of the poison on the pyramidal cells, metabolic changes also occur in the nerve cells as a direct result of the long standing anemia.

4. The blood vessels, pyramidal cells and the medullary fiber show similar degenerative changes at different levels of the central nervous system.

5. The foci of degeneration bear a definite and distinct relationship to the blood vessels.

6. In every case, the miliary foci of Preobrajensky were found in the pons.

7. Some of the nerve cells in every case with the exception of the third case, which was of very short duration, show diffuse pigmentation.

* It is quite possible that further investigation will prove that these psychoses are due to an encephalitis, and that therefore they belong in the group of encephalopsychoses rather than in the group of somatopsychoses.

8. In speaking of the neuropathology of pernicious anemia, it is not sufficient merely to describe the lesions found in the spinal cord. The brain changes are too numerous and definite to be omitted. The neuropathology of pernicious anemia should include the entire central nervous system.

I wish to express my thanks to Dr. Elmer E. Southard not only for placing the clinical material at my disposal, but also for his many helpful suggestions in carrying out this study. I also wish to thank Dr. M. M. Canavan for her kindly interest and capable supervision. These have been of inestimable value to me in the preparation of this paper. My thanks are also due to Miss E. R. Scott for her care in the preparation of the sections, and to Mr. H. W. Taylor for his care in the preparation of the photographs.

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Abstracts from Current Literature

L'HYSTERIE CHEZ LES PRISONNIERS DE GUERRE INTERNES
EN SUISSE. A. REPOND, Schweiz. Arch. f. Neurol. u. Psychiat. 3:
128, 1918.

An interesting point is contained in this article by an observer, apparently so placed as to have surveyed mental reactions under conditions that might specify further our notions of the psychogenesis of hysteria. He states that the Belgian and French prisoners of war, interned in Switzerland, have presented more frequently striking forms of hysteria, than have prisoners of these countries while held in German camps, quoting here from Birnbaum's articles.

The attacks were of the classic convulsive type with rigidities, excitement and amnesia. A considerable hysterical background was revealed in most by their impulsiveness, lability, suggestibility, hypochondrical notions, profound egocentricism and in many a pseudologia-fantastica. No traumatic causes could be isolated. As precipitating factors he saw the refusal of privileges, delays of letters, imposition of hard work, unsuccessful attempts at repatriation.

It may be that what is sifted down in the subjects of these observations are those definite hysterical types presenting in any given mass. What arrests the attention is the higher percentage of obvious hysterical disorders here than obtain in the camps of Germany. We are familiar with the type of conflict, defined by MacCurdy in the "Neuroses of War," as the most effective precipitator of the hysterical reactions; in these concepts the notion of the herd being given a nuclear position rather than the unconscious bases of the usual type.

The psychic factors contained in an internment camp are of course not strictly comparable to those with which MacCurdy had to do, for there is an apparent termination here of the immediate hazard of battle. From the Teutonic sources gathered by our author this is borne out in the relatively lesser number of the hysterical manifestations occurring there. Yet in the Swiss camp, where the step to freedom has been made, where repatriation is more nearly possible, the hysteria appears in its massive form. It looks as though the mechanism or the wish shrewdly has bent to the rigid and harsh reality of a German prison camp; but where the wish approaches as possible of fulfilment, at a site where repatriation may be gained, there is put under way in its most infantile and regressive form all the underlying movement of the unconscious.

The wishes sought to be gained are, however, not simply those involved in the freedom of the individual, but are discovered, for example, in a desire for the better quarters of the hospital, to which a malady may assign them, or to gain funds, or special attention. They appear frequently as threats to the officer if he failed to grant the privilege sought. Nor is the percentage of cure found here at all comparable to that which obtains at the front. The psychotherapeusis which succeeds best is one containing a promise of immediate advantage in case the malady immediately terminates. Those are cured who wish to remain cured.

One gets an illumination here of the value of the reality principle in the hands of those treating the neuroses at the front and assures one that beneath those conflicts whose solutions there have been so readily gained, there may remain concealed a characteristic infantilism to which under certain conditions the psychoneurotic appears always to regress.

PARKER, New York.

WHAT THE WAR HAS TAUGHT US ABOUT TETANUS. LOUIS BAZY, M.D., *Lancet*, 2:523 (Oct. 19) 1918.

As preventative, antitetanic serum has proved itself of very great value and the systematic administration of antiserum has proved wise. However, cases of tetanus have developed in spite of serum either (a) because the serum was insufficient to neutralize a large amount of toxin, or (b) because the immunity conferred was of insufficient duration. In the first case tetanus appeared early; in the second case, late. Clinically the late forms of postseric tetanus were of interest and unlike the usual picture. Frequently spasms were limited to the locality of infection; in some patients, partially immune, the bulbo-medullary centers remained unaffected, but symptoms referable to cortical stimulation (hallucinations, delusions, etc.) occurred.

For the prevention of early postseric tetanus the author recommends that from 20 to 30 c.c. of antitoxin be injected every eight days for one month, together with wide excision of bruised tissue. The serum, he believes, is best given by the subcutaneous route. For the prevention of late postseric tetanus vaccination is recommended. It has been shown that foreign bodies may harbor living tetanus bacilli for over 200 days, symptoms therefrom appearing after late operation or on commencing massage, or following some mechanical disturbance to nearby tissues. Cases in which late treatment—operative or manipulatory—is necessary, should not only receive repeated antitoxin injections as outlined, but should also receive tetanus vaccine. The vaccine he employs is the toxin used at the Pasteur Institute for immunizing horses. He gives this toxin iodized, subcutaneously, in successive doses of 4, 8 and 12 c.c. (1 c.c. of a 1:10,000 solution kills a guinea-pig of 400 gm.); he has seen no serious reactions in man.

By means of antiserum given at eight-day intervals for a month he feels that protection is afforded for forty-five days. By the subsequent use of vaccination he believes a much longer period of immunity is conferred. By combining the two in appropriate cases he is of the opinion that adequate protection against both early and late tetanus is afforded.

AYER, Boston.

THE EXISTENCE OF THE TWO DISTINCT PHYSIOLOGICAL SYSTEMS FOR THE TRANSMISSION OF MOTOR IMPULSES IN PERIPHERAL NERVES. J. RAMSAY HUNT, M.D., *Brain*, 41:302 (Nov.) 1918.

Hunt's studies have led him to formulate the hypothesis that the skeletal musculature is enervated by two distinct systems of motor fibers. One system is under the control of the corpus striatum and subserved by a strio-spino-neural system. Its function represents a primitive and diffuse form of motor activity characterized by automatic and associated movements. The system is termed paleo-kinetic. The other system or neo-kinetic system phylogenetically more recent is under cortical control and subserved by a cortico-spino-neural

system. The highly specialized motor function of this system transmits isolated synergic movements.

The previous studies of this writer on paralysis agitans indicate that the extrapyramidal motor system is under control of the corpus striatum and that loss of the large motor cells in this structure occasions a break in the strio-spinal system causing a progressive loss of certain automatic and associated movements. Paralysis agitans is the counterpart of the spastic paralysis following pyramidal tract lesions. The writer developing his theories continues this differentiation of motor pathways in peripheral nerves and differs with Sherrington in the conception of a final common pathway for motor impulses.

The writer draws an analogy between the two types of sensibility—epi-critic and protopathic—comparing these to neo-kinetic and paleo-kinetic motor functions, respectively. In restoration of function following gunshot injuries it would appear that the first evidences of motility are of the character of associated movements. Hunt relates the following case: A man, aged 30, was injured by a cut from a circular saw involving the musculospiral nerve in the middle of the arm. No improvement following after the lapse of one year, an operation was done, the nature of which was unknown to the patient and which was followed by some improvement, but the usefulness of the hand was seriously impaired. At the time of Hunt's examination fifteen years after the accident, there was a typical wrist drop with a total paralysis for all isolated synergic movements in the distribution of the musculospiral nerve including the supinator longus. When, however, the hand was closed, as in making a fist, the musculature of the entire forearm and upper arm was contracted—supinator longus, extensors, and the hand assumed a position of extension. This phenomenon was interpreted as an expression of automatic and associated motor function and of either restoration or preservation of the motor system of fibers conveying this function; whereas the other system of fibers—the neo-kinetic system—had been permanently interrupted. The difference in the behavior of the different systems may be explained by the assumption that the paleo-kinetic system is not only less vulnerable but regenerates more readily after injury. A further similar musculospiral case is reported by Hunt, also a published case of Oppenheim; and references are made to three additional cases illustrating clinically this same phenomenon in the distribution of other nerves, for example, the median-popliteal.

In the restoration of motility after facial palsy the writer believes that there is additional clinical confirmation of his thesis. Dependent on the location of the lesion there are recognized two principal types of facial palsy: one characterized by spasticity and loss of isolated movement (pyramidal tract lesion); the other producing rigidity and loss of the automatic and associated movements (pallidal system lesion). The expression of emotion is essentially of the automatic and associated type, and the emotional enervation of the face is centered in the corpus striatum. Voluntary motion is dependent on the integrity of the cortico-spinal system.

From the anatomic standpoint attention is called to the theory of dual enervation of striated muscles as enunciated by Botazzi, also to the presence of another type of end-plate in striated muscles demonstrated by Boeke which is the termination of a nonmedullated fiber and is apparently of sympathetic origin. These sympathetic endings enervate the sarcoplasm of muscle which is that component concerned with the slow tonic forms of muscular contractions and is plastic in its function. The quick contraction or twitch is the function of the anisotropic disk system supplied by medullated nerve fibers. The investiga-

tions of Langelaan and Sherrington show that these functions are entirely different from a physiology standpoint. It is believed by Hunt that certain differences in the function of the paleo-kinetic system and neo-kinetic system are closely related to this duality of function of the intrinsic muscle mechanism: notably, the twitch component is of more recent origin phylogenetically and has to do with isolated movements of cortical origin. Thus the anisotropic disk system could play a greater rôle than the sarcoplasm concerned with automatic and associated movements.

The studies of Ranson of nonmedullated nerve fibers in peripheral nerves, found in considerable numbers and thought by him to be concerned with the transmission of protopathic sensibility, are mentioned as suggesting and interesting similar study of motor fibers. Further, accepting the correctness of the theory which holds that in peripheral nerves there is a more highly differentiated form of nerve fiber subserving the neo-kinetic function one might expect a myelination of fibers subsequent to birth as in the case of the pyramidal tracts. The studies of Hatai along these lines in the anterior roots of the white rat, and of Boughton on the oculomotor nerves of the same animal show that the number of medullated fibers increases as the animal becomes older.

Large-sized and medium-sized medullated nerve fibers predominate in the ventral roots of the spinal cord supplying the muscles of the extremities which are concerned with finer cortical movements (in proportion of 5 to 1). On the other hand, finer nerve fibers preponderate in the thoracic and coccygeal regions in the proportion of 3 to 1. These anatomic facts taken from Quain's Anatomy are cited in confirmation of the theory under consideration.

Returning to the clinical aspect of the subject, the two systems are related to the classic types of central hypertonus-spasticity and rigidity. The removal of inhibitory control over the spinal centers would result in these manifestations of hypertonus. Spasticity results from the removal of cortical inhibition and evidences the altered function of the neo-kinetic system through the anisotropic disk system. Spasticity is characterized clinically by the more elastic quality of the tonus, exaggerated tendon reflexes and clonus indicating a greater development of the contractile element of motility. Rigidity results from the removal of inhibition of the corpus striatum and evidences the altered function of the paleo-kinetic system through the sarcoplasm. It is characterized by a more uniform increase in tonus, is of a more waxy character, lacks the so-called clasp-knife tension of spasticity, and clonus and exaggeration reflexes are absent. Rigidity is typified in paralysis agitans, the isolated movements of cortical origin being merely masked. Attention is called to the deformity which is so characteristic in pyramidal tract lesions (Wernicke-Mann Type). The paralysis is one of neo-kinetic motility and for the reason that the extremities and chiefly their distal portions are affected, as this portion of the musculature represents the highest degree of specialization of cortical movements. Contrasted with this type, the deformity of paralysis agitans is concerned chiefly with the trunk and root segments and the paralysis of this disease is one of automatic and associated activities of muscles.

Consideration of the reflex activities in the two different central types of palsy brings out noteworthy differences when either the cortico-spinal or strio-spinal systems are involved. In paralysis agitans or an affection of the paleo-kinetic system the skin reflexes are unchanged as they depend on reflex activity of a higher type (afferents, epicritic sensory system, efferents neo-kinetic system). Also the tendon reflex show no special abnormalities, nor is clonus present. In spastic paralysis due to lesions of the cortico-spinal

system, the abdominal and cremasteric reflexes are abolished. The Babinski reflex which is of striate or strio-spinal origin replaces the normal plantar reflex. Striking is the presence of exaggerated tendon and periosteal reflexes and clonus.

Finally the cerebellum is considered as consisting of a paleo-cerebellum controlling the postural or static function of automatic and associated movements and a neo-cerebellum for the postural control of isolated synergic movements of cortical origin. These stand in relationship with the strio-spinal system and to the cortico-spinal system.

This important article of Hunt is worthy of analysis in that it is an attempt to correlate much that is recent and important growing out of numerous studies of the motor mechanisms of the nervous system. The author, by further developing his ideas concerning the function of the striate body, brings in review his wide and intimate knowledge of motor function to support his hypothesis. But, as the author himself admits at the beginning of his article, the theory is more suggestive than complete. It is founded more on clinical interpretation and theoretical considerations rather than on anatomic or physiologic facts or experimental evidence. The theory would appear to stand or fall on the existence of an anatomically distinct system of fibers in peripheral nerves centrally controlled by the striate body, with spinal and reflex connections and finally ending in a separate system of motor endings in skeletal muscles. Considered anatomically the evidence in support of his theory appears to be based on the demonstration of sympathetic nerve endings in striated muscles and the suggestion that the peripheral nerve connections might be demonstrated in some such manner as Ranson has demonstrated afferent nonmedullated fibers. It would thus appear that the strio-spino-neural system is in part at least sympathetic, yet this is not clearly brought out by the writer. Associated and automatic movements, function of the paleo-kinetic system would be a manifestation of sympathetic nerve function, under the control of the striate body. This would be the logical deduction if we follow correctly the line of argument. That the rigidity of paralysis agitans might be due to disturbed function (tonus) of the peripheral sympathetic system due to removal of central inhibition allowing fuller play of the plastic component due to sarcoplasmic activity is conceivable. There is some confirmation in this view perhaps by the rigidity observed by Sherrington in decerebrate animals, and believed by Langelaan to be due at least in part to a spasm of the sarcoplasmic part of the muscle substance. But that this same mechanism would also serve for automatic and associated movements appears to us to be too all-inclusive and especially as it is argued that loss of central control over the paleo-kinetic system would abolish these movements. In other words, removal of inhibition of the paleo-kinetic function would, on the one hand, cause hypertonus—a positive mechanism—and, on the other hand, abolish automatic and associated movements—also a positive mechanism.

Hunt believes that the twitch component concerned in muscular contraction and related to the anisotropic disk system has to do with isolated movements of cortical origin and belongs to the neo-kinetic system. Recent studies of tonus and reflex activity in uncomplicated cases of complete division of the spinal cord are not in accord with this view. For example, Riddoch observes in these cases an absence of the plastic component and a retention of the contractile element in the muscles. The patellar reflex is brusque and followed by a quicker relaxation phase as compared with cases of incomplete division that is, in the sense of Langelaan that the slow tonic component,

not the twitch component, is altered by severance from cortical control. It may be said, however, in this connection that in a case of complete cord section reported by Schaller the muscle tonus was normal or increased.

It is assumed that the paleo-kinetic system is less vulnerable to injury or regenerates more rapidly after injury thus explaining the first return of automatic and associated movements when function is restored. The cerebellum is divided into a paleo-cerebellum and neo-cerebellum for postural control of the automatic and associated movements, on the one hand, and isolated and synergic movements, on the other hand, without a clear exposition of the mechanism of these relationships. We thus discern a tendency to reason from this attractive hypothesis rather than to build an argument from accepted ideas of anatomy and physiology. Before we can accept Hunt's hypothesis we must have a more convincing demonstration of separate pathways for motor fibers in peripheral nerves.

SCHALLER, San Francisco.

WHY IS THE "UNCONSCIOUS" UNCONSCIOUS? MAURICE NICOLL,
W. H. R. RIVERS AND ERNEST JONES, *Brit. J. Psychol.* 9:230-247 (Oct.)
1918.

Rivers, after a delimitation of "unconsciousness," emphasizes this by a common instance of an experience, unconscious for thirty years, yet related to a persistent and marked symptom, reappearing in consciousness at the end of this period. "This active existence, apart from experience readily accessible to consciousness, is known as dissociation." His question are, "why experience becomes unconscious and why, having become unconscious, it should persist in this dissociated state. . . ." Rivers locates the solution in the life of the organism. He describes in the butterfly the formation in its larval stage "of a highly complex body of experience, based on a certain mode of progression and certain methods of obtaining and utilizing food." "So far as experience is conscious we suppose it takes part in the regulation and success of the life of the animal." He now sees, in the persistence in the consciousness of this experience into the next stage of existence, the liability of that which would impair those movements adapted to a now totally different mode of existence. "The perfectly adjusted flight of the butterfly could only be disturbed if memories derived from the larval state of existence entered into the consciousness."

He makes these examples of what less strikingly takes place in every animal, including man. A similar interference is seen in the more evolved organism through any persistence of earlier unmodified modes of consciousness. "The diversity of the different phases of the life history and the incompatibility of the reactions of one phase with another" is the reason why the conscious states become unconscious. He further sees this suppression or dissociation as peculiarly complete in those organisms where there is absolute dependence of the animal on instincts. Its high point in man occurs also with those years where the conduct depends largely on inherited impulses directly related to the primary needs of life. "It becomes a question whether the dissociation of the mental life of Man is not a mode of reaction belonging originally to its instinctive aspect which has continued to be utilized after this aspect has been largely replaced by intelligence or reason. Dissociation would thus be a state especially prone to come into existence whenever it is required to put instinctive modes of reaction into abeyance, to suppress instinctive modes of behavior which would interfere with the harmony of an existence based on less immediate and more modifiable reactions."

Yet the biologic mode of dissociation he notes as having been modified, for, obviously, affective responses, as of the infant, appear ready to present if later conditions call them forth. In place of the complete suppression there comes into existence a mechanism whereby certain parts which were useful could be utilized, while others were dissociated. He sees this principle permeating the whole of evolution. The process of repression of the psychologist corresponds exactly to the inhibition of the physiologist. "Nature has always utilized existing structures and functions, combining the new with the old, utilizing what she needs and inhibiting those parts of the products which would interfere with the normal action of the new. The symptomatology of disorders of the nervous system mainly depends on the coming to the surface under pathologic conditions of those older and cruder activities which have been controlled or suppressed by the later products of evolution."

Thus seen, repression (for here is the core of largest use in this discussion) is something close to an instinct; its function is to put out of the running other instinctive modes of behavior which interfere with the mechanism made up of a combination of instinctive with intelligent modes of reaction. This makes, as Rivers says, the unconscious the home of the instincts. With this endeavor to discover some "biological archetype" for the processes of suppression and repression, Jones immediately expresses an approval. He perhaps is right in holding Rivers' examples as over-remote. But his shrewdest and broadest critique lies in his statement that, "in the endeavor to establish a purely utilitarian basis for the exclusion of unconscious mental processes, Dr. Rivers has been too much influenced by the English biologic doctrines of the nineteenth century, which, as is now known, interpreted all vital processes too exclusively in terms of survival value." Aside from the difficulty in bringing the phenomena under this head of utility values, he properly defines our necessity for stating them in a preliminary way in terms of affects. What Rivers called the incompatibility between instinct and intelligence, Jones relates as of a hedonic nature. Yet there is a definite aim here on Jones' part in considering the possibility of an approximation of views in the examination of the ultimate sources of repression. In approaching this he develops in a clear form the notion of Freud's primary and secondary systems, a notion here described as avowedly tentative.

Looking at the wish—a "gigantic reflex"—as, in the mental sphere, the expression characteristic of all activity to restore a disturbed equilibrium, he pursues its route in the primary system with the later initiated activity of the secondary system coming on the heels of the "bitter experience of the inadequacy of the first hallucinatory gratification." The second inhibits the movement of physical energy; the primary allows the freest possible movement throughout the entire system; whatever is disagreeable is ignored, for only the fulfilment of the wishes is permitted. This latter course is not possible for the secondary system. In this theory of Freud's, with which the reader is familiar, Jones sees, in the interrelation of the two systems, that which falls in well with Rivers' utilitarian principle, for the gain to the organism is evident in the subjection to the reality principle belonging to the secondary system of the hedonic principle appertaining to the primary. There is here a guidance and inhibition of one kind of activity by another which in evolution has come to be superimposed. Probably, also, Jones defines the reality principle in its evolving position as functioning to extend and elaborate the pleasure principle. This he emphasizes as something over and beyond its functioning as a utilitarian value.

A difference in concept appears at this point. The examination of ultimate sources has given a considerable agreement between the biologic notion of Rivers and the expressions of the Freudian theory of the mental systems. Perhaps the difference is a bit fictitious and necessitated by the guiding lines of conscious and unconscious, necessitated by the discussion. The two systems are viewed as the precursors of the unconscious and conscious mind of later life. The point here is not strong as, in Jones' statement, "the mind of the infant can hardly be called either unconscious or conscious, though probably it is nearer to the former." Certainly the notions of the primary and secondary are to be read in the relation of conscious and unconscious, to the benefit of the latter term, as in his defining of the inadequacy of the secondary system at all times to inhibit and control due to its rapid growth and changes, resulting in a sharper contrast between the instinctive or infantile and the higher standards of the later secondary system. Yet in the point of the secondary system serving the primary, though controlling it, appears the presence of the wish and the hedonic quality present. That he places this at a later point than the "utilitarian" function, is to be noted and approved. The arresting fact lies in the extension of the concept of repression beyond that usually connoted. A biologic connection is here suggested of considerable heuristic value.

Both Rivers and Jones are rather severe in their consideration of Jung's theories as presented by Captain Nicoll. The notion of the unconscious as an inheritance, a racial background of the mind, as a collective unconscious with the personal unconscious, "an excerpt of the collective containing repressed material that has an intimate personal significance," this naturally would fall across the line of both his fellows in the discussion. "The unconscious is unconscious because it is not yet fully adapted to reality." Jones remarks on this view as to its being not yet adapted, that, from Captain Nicoll's lofty conception of the nature of the unconscious, one might expect that it easily would be. Also he refutes, in his critique, the notion imputed to Freud that all unconscious processes have at one time been in the individual's consciousness. "The sources of conflict lie in the unconscious; here are forces of progression as well as forces of regression": these statements of Nicoll give the whole notion of evolution a location, a special site, in consciousness. While Rivers sees in the unconscious a storehouse of instinctive activity, more or less incompatible with later products of evolution, Nicoll, after Jung, sees "an inexhaustible reservoir with which man is provided at birth and from which he can draw material for his psychic development." Yet this peculiar diversion of concept throws into clearer relief the essential qualities of the discussion, earlier presented, wherein both the ideas of the unconscious have been defined through the notions of the dynamic mechanisms of repression, while the latter itself has gained considerably in depth of background.

PARKER, New York.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

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WALTER TIMME, M.D., *President*

BILATERAL FRONTAL HEMORRHAGE WITH MENTAL AND PHYSICAL SIGNS PRECEDING AND ACCOMPANYING THE HEMORRHAGE. Presented by DR. FREDERICK J. FARNELL, Providence, R. I.

This case was that of a woman, aged 49, whose make-up, as far as could be ascertained, had always been considered normal. With a clear history of no previous attacks or upsets she developed a mental state not unlike the manic form of manic-depressive insanity, which settled, after several months, into a hypomanic condition. Physically there was hypertension; blood pressure 210 systolic and albumin in the urine. Approximately twelve months after the onset of the so-called functional psychosis, she had a cerebral hemorrhage with clinical signs suggesting the intraventricular type. This was followed by a complete change in the mental picture. From a functional psychosis with thought (thinking) disorder, she settled into an organic psychosis with an obstructive disorder, or an intellectual loss. The physical state then improved and she was soon up and about, but there was no change whatever mentally. In five or six weeks signs of cerebral compression developed and ultimately contributed toward her death.

The interesting features might be grouped as (a) mental and (b) physical, in an individual who developed two psychoses. Her first aberration was characterized by emotional elevation, acuity of emotional response and quick recognition of logical judgment and even, at times, critical judgment. Following hemorrhage this emotional state changed entirely into an actual loss with those accompanying behavioristic traits which so characterize the personality of organic brain disease; deterioration in feeling, instinct, conduct, etc. Then, too, such organic functions as intelligence, judgment, insight, etc., which in her primary psychosis were only interfered with to the extent of thought disorder, too many thoughts coming at once were practically completely destroyed in her second psychosis.

The initial physical disorder was limited to hypertension and albumin in the urine. The second physical disorder was considered as a symptom-complex related to the ventricles. The third physical complex was a syndrome indicating cerebral compression. Pathologically the brain (which was exhibited) showed bilateral mid-frontal hemorrhage with organization, secondary necrosis and softening of the brain tissue itself.

These facts were offered for consideration, especially so to those whose interest lay in the field of psychopathology with special reference to personality and psychosis, and in addition, to the histo-pathologist to whom interest would be manifest in the fact that the lesion was bilateral and the vascular sclerosis localized in the cerebral vessel system.

DISCUSSION

DR. SMITH ELY JELLIFFE asked if Dr. Farnell might have something to say about the hypertension states with increased blood pressure and the relationship of their emotional causes to cerebral hemorrhage. In one of the discussions on psychoanalysis held before the Neurological Society recently, the speaker presented the situation of a case of autonomic responses to unconscious emotional stimuli, and spoke of the tendency of these cases to develop these cerebral hemorrhages. He said that the psychotherapeutic mode of approach could be very successfully pushed with the hope of reducing the hypertensive state by getting at the emotional causes for it. He had taken occasion at that time to discuss rather briefly what some of the problems were and related the partial history of a patient who had a blood pressure of 235 systolic, with almost *petit mal* lapses, who was correctly diagnosed by an internist as a nephritic and given six months to live. What it was that lay behind the clinical picture was not discovered nor even sought for by the internist who made this diagnosis, the dynamic reasons for the hypertension nephritis. When the dreams were investigated the cause for the distress of the patient was quite apparent; the unconscious was engaged in a sadistic attempt to destroy everything about her and the result was that she was destroying herself. If it had kept up, she might have died, if not of nephritis, of a cerebral hemorrhage as had Dr. Farnell's patient. The psychoanalytic mode of approach offered a dynamic pathology which was not in the usual ken of the internist and proved its value in these nephritides with their disturbed emotional undercurrent as the real dynamics of the situation.

DR. I. ABRAHAMSON said he would like to know more about the behavior of the reflexes. That all reflexes were absent after the bilateral hemorrhage in both ventricles he knew, but it was not clear to him whether they returned after the condition cleared up and, if so, the order of their return.

DR. CLARENCE O. CHENEY questioned whether the hemorrhage was of the intraventricular type, for the specimen did not seem to show evidence of a breaking of the ventricular wall; it looked rather as if the ventricles had been compressed by the hemorrhage. The absence of blood in the ventricles would have accounted for the absence of blood in the spinal fluid. He recalled one case necropsied several years ago which showed intraventricular hemorrhage causing sudden death and believed that if a hemorrhage of the size demonstrated in Dr. Farnell's specimen had entered the ventricles immediate death would have resulted. The distribution of the hemorrhage suggested that it had originated from one of the anterior cerebral arteries.

DR. FARNELL, in answering the discussion, replied to Dr. Jelliffe's question regarding the hypertension that, although the patient had a blood pressure of 210 systolic with albumin in the urine on admission to the hospital, the albumin soon disappeared. The hypertension continued until after the hemorrhage, and from that time until death the blood pressure was below 160 most of the time. With reference to the emotional factor and its responsibility for hypertension and nephritis, it was impossible to do an analysis of the patient, for even when she settled into the hypomanic condition she was beyond reach of questions and psychic control. Answering Dr. Abrahamson's question as to the reflexes, they returned and became exaggerated, that is, all deep reflexes became exaggerated. The superficial reflexes returned and even when she was up and about she had exaggeration of reflexes, double ankle clonus and double dorsal extension of the great toes. Ten days before death she was

again minus all reflexes. She was in a deep stupor with signs of cerebral pressure. Muscular flaccidity with abolition of reflexes was again marked, and the condition then and at the time of the hemorrhage was noted by all the men who examined her. The physiology could not be explained beyond its being a question of first hemorrhage and pressure, and second organization and pressure. The clinical diagnosis made at the time she had the hemorrhage was bilateral ventricular hemorrhage, at least, the symptom-complex suggested that, though there was no blood in the cerebrospinal fluid. That might seem impossible, but for two years the speaker had been examining the spinal fluid of cases of cerebrospinal meningitis, and it was not uncommon for this syndrome to give intraventricular symptoms from accumulation in the ventricles of fluid or obstruction to the flow.

THE TONUS OF AUTONOMIC SEGMENTS AS THE CAUSE OF ABNORMAL BEHAVIOR. Presented by DR. EDWARD J. KEMPF of St. Elizabeth's Hospital, Washington, D. C.

DR. KEMPF (by invitation) delivered this address in which he traced the causes of abnormal behavior to physiologic facts. The evolution of man from the lower biologic types was sufficient reason for giving the primitive autonomic apparatus the greatest emphasis in formulating a conception of the personality. The lowest biologic organisms have a relatively highly developed autonomic apparatus, but a poorly proficient apparatus as the instrument by which the autonomic apparatus masters its environment. The old assumption that "the brain is the organ of the mind" is entirely unsatisfactory for localizing or explaining the dynamic forces that make up the personality.

The autonomic apparatus, as herein conceived, is constituted of the digestive, circulatory, respiratory and urinary system, the glands of internal and external secretion and their ganglionic nervous systems; that is, the ganglionic nervous systems lying outside of the brain and spinal cord and those ganglionic types of centers embedded in the brain stem and spinal cord. It is obvious that this is the apparatus that regulates the accumulation and assimilation of energetic products from the environment, regulates their transformation, distribution and use, and the elimination of the waste products. It might be said that these processes constitute about all the fundamental functions of living, and that the striped muscle apparatus and its cerebrospinal nervous system had been developed in order to obtain the necessary means from the environment.

The physiologic researches of Cannon and Carlson on the peripheral origin, in the stomach, of the craving for food, as a typical acquisitive-assimilative compulsion, and Mosso's and Pellacani's experiments on the postural tonus of the bladder, show that when the grip of the bladder wall on the inert contents raises the pressure to over 18 c.c. a type of localized itching is aroused which constitutes the desire or craving to urinate, and which, as it becomes vigorous, compels the organism to behave so as to relieve the hypertension of this segment. This may be considered to be a very typical emissive-avertive type of compulsion. All compulsions to act are either acquisitive or avertive in relation to the environment.

Freud's suggestion, that all emotions and sentiments are really cravings, is further borne out by the studies of Cannon and others on the physiologic changes that occur when the individual feels "fear" or is said to be "afraid." Since certain types of gastric contractions cause the intragastric itching felt

as hunger, it is consistent to consider that other changes in the gastric functions, such as diminution of peristaltic functions and the maintenance of spastic tensions, when the individual is exposed to some type of actually injurious or potentially injurious stimulus, arouse an afferent stream that is more or less painful and disagreeable—that is to say, fearful. It is obvious that only those primeval animals or rather autonomic systems that felt distressing, fearful tensions, sought to protect themselves and so, by surviving in the struggle for life, have transmitted this fundamental attribute or function to man.

Wertheimer's experiments on the unconscious anesthetized dog, in which he injured the sciatic nerve in a manner that would surely cause pain in a conscious animal, shows that gastric changes occur which are very similar to the fear producing tensions, without the faculty of perception, to arouse the emotion, being present. This shows that at least certain cerebral integrative activities which enable peripheral activities to coalesce into perceptual images (or thoughts) are not necessary to cause many of those definite, important, autonomic tensions which, if the animal were conscious, would certainly cause it to be aware of very disagreeable (fearful) visceral feelings.

Like *fear* the other primary affective cravings, such as *anger, love, shame, disgust and sorrow* have their origin in characteristic peripheral disturbances in various visceral segments, and these peripheral disturbances are constituted of changes in the muscular activities, particularly the tensions of the viscera, stimulating the local sense organs. This means that it is of the utmost importance to recognize that our affections are symptoms of autonomic tensions and activities and we must practice visualizing these activities behind the symptoms that we see or hear complained of.

These autonomic-affective tensions set up afferent streams of nerve impulses which, as the autonomic component (Langelaan), contribute greatly to regulating and sustaining the postural tension of the striped muscle apparatus, and the tension of the striped muscles in turn stimulate the proprioceptors embedded in the muscles and tendons and about the joints, setting up converging kinesiastic streams which coalesce into images and concepts, that is, the mental pictures constituting most of the content of consciousness. In a sense we think with our muscles.

The present controversy between Langelaan, de Boer, von Rinjberk and J. G. Duesser de Barenne as to the exact manner and through what channels this influence is exerted is not so important for psychology and psychiatry as the fact that it does occur in some *quick, intimate* manner and follows the law of the autonomic-affective apparatus striving to maintain a state of comfortable tension with the greatest economy of extent and duration of effort. This law may be formulated as follows:

As the autonomic-affective apparatus is forced into a state of unrest, either through metabolism or endogenous or exogenous stimuli, it compels the propiicent apparatus to adjust the receptors in the environment so as to acquire stimuli that have the capacity to produce comfortable postural readjustments in the autonomic apparatus. For example, when the autonomic apparatus of a child assumes fearful tensions because of the barking of a dog the affect from these tensions compels the child to run to its mother who, as a soothing stimulus, readjusts the tensions. So too, the business man takes out insurance as the soothing stimulus, the fearful sinner goes to church, the savage and the modern speculator wear charms and fetiches, in order to counteract the fearful stimulus existing in his expectation of a disastrous fire, storm or coincidence.

Von Bechterew has shown that various autonomic segments and even the simple striped muscle reflexes become conditioned by experience to react to certain stimuli. This occurs by the reflex being aroused by the primary stimulus while it is associated concomitantly with other stimuli which ordinarily have no effect, but which, after repeated simultaneous association with the primary stimulus, come to have the same influence on the reflex that the primary stimulus had. For example, when a child going barefooted for the first time in the grass, steps on a bee which stings its foot, the child, for some time after this experience, has strong autonomic fear reactions which prevent it from walking on the grass while barefoot. Here then the grass, formerly a pleasant stimulus to the bare feet, by being associated with the bee sting comes to have the capacity to arouse strong autonomic fear reactions. It becomes a painful stimulus, while to other children it is a pleasant stimulus. We can readily see how, by experience, the individual segments of the autonomic apparatus of an individual becomes conditioned to react to stimuli that have little or no effect on other people and determines most of our eccentric or individualistic preferences and prejudices, our "taste," hobbies, phobias, obsessions, compulsions, vocational pursuits, etc. No doubt all our selections and aversions for simple things and for complicated things, that are immediately present or that may arise in the future, are greatly determined by your autonomic-affective cravings having been conditioned by previous painful or pleasant experiences to seek or avoid the future possibility.

Since all the autonomic segments must obtain their stimuli through the proper exposure of the favorite receptors, for which they have become conditioned, there is an incessant convergence on and striving for control of the final common motor paths, and our complicated stream of thought and overt behavior must be seen as the resultant of these converging forces. When any particular autonomic segment becomes hyperactive and tends to dominate the autonomic union and obtain control of the striped muscle apparatus the individual becomes conscious of a definite stream of thought which is symptomatic of the activity (as gastric itching—hunger—thoughts and acts about when, where and how to get food; cystic itching—craving to urinate; reversed gastric and esophageal peristalsis and feelings of nausea with aversive compulsions for a particular odor, vision, taste, person, or suggestion).

It follows logically that if one autonomic segment becomes thoroughly conditioned to react in a distressing manner to certain stimuli, and other autonomic segments become thoroughly conditioned to react in a pleasant manner to certain stimuli, whenever the individual happens to meet those two groups of stimuli associated together in a situation, he will feel a confusion of tensions with compulsions to seek the advantages of the situation as well as compulsions to avoid it. For example, a young married man complained that although he was fond of his wife and desired to be loyal and faithful to her, that "such asinine things" as the hair on her legs caused him to lose his sexual excitement (depression of the tonus of an autonomic segment) which irritated him exceedingly. Many of the attributes of his wife, such as her wit, sense of humor, facial expression and coyness, as stimuli had a decidedly invigorating effect, but when he made further approaches he met with a stimulus that had quite the opposite effect. He finally compelled his wife to shave her legs in order to remove the distressing stimulus. We see here how the autonomic apparatus—looking at it in a biologic sense—compels the love object to remove or avoid stimuli that jeopardizes its potency as well as seek stimuli that tone up the autonomic segment.

At birth we have a perfectly organized but unconditioned autonomic apparatus with a very poorly coordinated projicient apparatus. The autonomic apparatus begins immediately to organize the projicient apparatus to suit its cravings in their struggle with the environment, and we see this process continuing throughout life as the individual develops his education, vocation or profession. For a considerable period after birth the infant indulges *heedlessly* in its segmental pleasures, such as nursing, urinating, defecating, cooing and screaming, without regard to the interests of other people. But these indulgences soon become an imposition on many of the autonomic interests of its parents and its social group, and they in turn are compelled to exert an incessant pressure on the infant which eventually conditions and more or less conventionalizes its methods for acquiring gratification for its segmental pleasures. Thus the infant gradually becomes conditioned to avoid the loss of favor and esteem of its parents and playmates because, when in disfavor, it is subject to many distresses, such as physical punishment, humiliation, lack of petting, feeding, etc. On the other hand, by behaving in a manner that wins favor and esteem from its associates, many of its segmental cravings are more easily gratified, such as cravings to be petted, played with, fed, given preferences. Gradually we see the infant changing from heedlessly enjoying its segmental pleasures to secretly doing so, such as nocturnal bed wetting. Then as the *ego* develops, the coordinations to control them entirely, in order to prevent the loss of esteem, become apparent. That is, the segments of the autonomic apparatus which are similarly conditioned gradually become integrated into a unity to prevent any hyperactive segments from jeopardizing them. This process of integrating into a unity is a *compensatory* reaction to prevent getting into the *fear, shame, sorrow* or *anger* state; autonomic compensation being one of the most fundamental attributes of living tissue.

The development of the *ego* begins as soon as the infant begins to fear to lose the favor and esteem of its comforters and protectors by becoming inferior (organically or functionally) to a competitor, or by self-indulgently yielding to oral, anal, urethral, etc., pleasures, by sucking, defecating, urinating, screaming, stealing, lying, etc., without regard for the feelings of others.

Any form of fear or pain no matter how mild or indirect the cause, initiates more or less of an autonomic compensatory reaction; hence, the individual's incessant compensatory striving to learn to help and improve itself is really *the autonomic apparatus striving to avoid getting into the malnutritional fear, shame, or sorrow state*. In due time this incessant striving, to avoid the stream of incessant interrelated fear-causes that confront the child during the day, becomes knitted or integrated into a complicated unity that eventually comes to regard itself as "I," or the *ego* and its various segments (teeth, eyes, stomach, etc.) as "mine." Now the perversely conditioned segments that jeopardize the *ego* become outlawed as "not mine," or sinful, evil, the devil, "hypnotic influences," "secret forces," etc.

Serious and fatal interautonomic conflicts occur when most of the apparatus is conditioned to strive for biologically and socially estimable things and one or more vigorous segments becomes intolerably or *perversely* conditioned. This is the foundation of the anxiety neuroses, the benign and pernicious psychoses and many forms of criminal or asocial adjustments. This constitutes the conflict between the *ego* and the *not-ego*, that is, the struggle between the autonomic apparatus, coordinated into an egoistic unity or personality, striving to win social esteem, and the self-indulgent segmental crav-

ings that only crave for the counter stimulation that neutralizes or gratifies their tensions; as in masturbation, sex perversions, envy, gluttony, slothfulness, etc.

The *ego* can only control the jeopardizing segment by preventing it, more or less, from using the final-common-motor-path or striped muscle apparatus to acquire what it needs. When the jeopardizing craving is permitted to cause the *ego* to be conscious of its needs but is not allowed to act, it is *suppressed*, and when it is also prevented from causing consciousness of its needs, it is *repressed*. The suppressed and repressed hypertense segments, like compressed springs, exert an incessant, severe pressure to break through the resistance and obtain gratifying stimuli. This is shown in sudden changes of purpose, selections, obsessive thoughts, errors, accidents, misinterpretations, dreams, delusions, hallucinations, mannerisms, old memories, deliria, etc. By a *summation* of the repressed cravings, or the fatigue or weakening of the repressing *ego*, a dissociation of the autonomic apparatus or personality occurs and the *ego* is forced to struggle with all sorts of compulsions, delusions, hallucinations, etc.

Here then lies the psychopathic struggle. The fear of the loss of social esteem initiates the compensatory striving, which, because of the vigor of the fear of the influence of the repressed, tense, autonomic segment, becomes progressively eccentric, finally causing the loss of the confidence and esteem of the social group. Now a vicious affective circle is established which tends to eventually destroy the socializing capacities of the personality. Gradually as the *ego* becomes more and more asocial the erotic segment obtains complete control of the personality.

A photograph of the perpetual motion machine of a paranoid negro was shown to illustrate how fear of the loss of sexual potency and of becoming an oral erotic homosexual initiated the eccentric, compensatory, divine inspiration (a truly biologic compensation) to build a "perpetual motion machine" which would make him a great prophet, allow him to found a faith, have many wives, etc. This perpetual motion machine is called the "first church" . . . "where the blood of the world is mixed," and is a simple copulation fetish.

Photographs of so-called hebephrenic dementia praecox cases showed the women squatting like apes and the incessant attention and frequency with which their hands counterirritated the urethral, anal and vaginal zones showed how, as biologic types, the anal and genital autonomic segments had destroyed the *ego* and dominated the autonomic apparatus.

Another photograph was shown of a soldier who carried his foot over his anus. He had passed through an anal erotic homosexual panic in which his delusions and hallucinations of being assaulted were caused by the anal erotic cravings (like gastric cravings and thoughts of food) seeking appropriate stimuli and his defense against the compulsion was a violent functional distortion compelled by the autonomic apparatus, as a compensation, in order to protect itself from going into the fearful state. He anxiously protested that he would go "mad" if the leg was straightened out.

DISCUSSION

DR. SMITH ELY JELLIFFE said that the point of view presented by Dr. Kempf struck a responsive chord in his own thoughts, and he had certainly performed a signal service in that he had bridged successfully two parallel lines of interpretation which were found throughout the medical community.

There were those who held that mental causes and somatic causes ought to be considered as separate types of activity. Not only through this presentation, but through Dr. Kempf's work on "The Autonomic Factors in Personality," these two parallel trends had been made to merge and a synthesis was presented whereby one could understand the individual working as a unit. What Dr. Jelliffe had to say he would confine to a few points, one of which interested him a great deal because it so frequently came up as a point of issue between these apparently parallel types—the question of infections, auto-intoxications and focal infections. Perhaps some of those present would recall an illustration of this, a patient who having suffered from a severe compulsion of washing the hands came for treatment at the age of 52, the compulsion having existed since she was 18 or 19 years of age. In the early days of the compulsion the washing of the hands was associated with certain prayers for purity, cleanliness and holiness, and she got along very well, the personality adjusting itself. At the age of 23, however, she had an attack of influenza and immediately the depressing effect of the influenza toxin broke down the adjustment of the hand washing and the prayers and she developed two new symptoms—diarrhea and auditory hallucinations in which obscene voices made vulgar references to defecation. If one conceived of an individual having 65 per cent. efficiency and a loss of 35 per cent. in a range of 100, the handwashing and prayers took up the 35 per cent. of the loss under ordinary circumstances, but when the influenza toxin came along, 10 per cent. more load was drawn on the autonomic segment adjustment, the diarrhea representing 5 per cent. and the hallucinatory projection representing 5 per cent. As a result of the strain she made a suicidal attempt. Ten years later another attack of influenza produced a similar result with diarrhea and hallucinatory voices which took on a more erotic form. Later, at the age of 52, a partial analysis enabled the patient to partially understand the compulsions as anal-erotic components in the personality. In an attack of influenza following the analysis she developed a diarrhea, but did not hear the voices and made no more attempts at suicide. At 52, she was much better able to handle her autonomic segment maladjustment thrown out of gear by reason of the toxin of influenza.

It was not influenza toxins, nor poisons from the intestines, nor infected tonsils that produced these situations in so many, but they should be considered as partly accessory in the breaking down of the combination. The real difficulty was the personality difficulty and the infection or toxemia was an additional factor to that difficulty. Furthermore, Dr. Jelliffe said that he thought that the original personality difficulty, if it could be analyzed out into its original roots, would afford a clue as to reasons why the infection or the toxemia could localize its effects in certain autonomic segments. Such autonomic segment neural pathways were under constant tonal maladjustment; they were overactive in the attempt to effect healthy functioning in the segment in spite of the instinct distortions, due to the conditioned segment stimulus to early emotional stimuli. After many years of such faulty strain in the reciprocal activity of the two components in the vegetative arc, the resistance of the tissues under the neural innervation in this arc were so seriously undermined as to permit of localization of an infecting or toxic agent in that arc. This was a local anaphylactic sensitization. Thus the localization of the disease in the badly functioned autonomic segment.

Dr. Jelliffe spoke of the influence of faulty anal neurotic stimuli, under constant repression, and the possible determination of rectal crises in tabes, localizing themselves as rectal rather than other types of crises because of

this autonomic struggle. An illustration was cited in which a partial uncovering of a passive pederastic unconscious repression in a tabetic with rectal crises possibly precipitated an intense delirium with fecal content. The speaker said he was simply following the clue which Potzl, Adler and others had offered in their attempt to answer important questions concerning why one or another organ or special parts of an organ were involved in a diseased process.

DR. ADOLPH STERN considered Dr. Kempf's presentation especially interesting from the point of view that, as suggested by Dr. Jelliffe, it served to bridge over two apparently conflicting conceptions of mental (emotional) processes, namely, the physical and psychologic, by giving the physiology of the emotions. This conception of Dr. Kempf, explained in concrete form, gave the physical basis for various (neurotic and psychotic) symptoms, which were in a measure, understood by psycho-analysts and explained by them as physical manifestations of thought processes. These now had a physiologic explanation as well.

It was also very instructive to find that the laboratory experiments on animals by Cannon, Crile, Sherrington, and others served to establish on a firm foundation the concept of the unconscious or autonomic system as presented by Dr. Kempf. Cannon especially has demonstrated the bodily changes that accompany the strong emotions and the instinctive cravings. Judging from the sensations as described by patients suffering from neurotic symptoms in the form of bodily sensations, that is, various paresthesias, etc., it was now known that such sensations were not at all "imaginary," but that they had a definite physical, physiologic basis consisting in a change, in the region in which the sensations were felt, from the normal tonus maintained by the autonomic system, the change being one determined by the emotions present at the moment.

Referring to the "condition reflex" and its bearing on psycho-analysis, a certain patient bore for many years a more or less conscious hatred for his father. He was always very irritable and easily incensed at any attempt to give him an order, even though he complied readily enough. He was very ambitious, the ambition being in a great measure a desire to be superior to his associates. Failure in any ambition was always accompanied by anger at the successful individual, whose ability he depreciated. This patient was aware of a general sense of tension of all muscles of the body, especially the arms and face. He frequently, without any conscious cause, doubled his fist and smote the palm of his other hand. On analysis he disclosed that the peculiarities noted were determined (conditioned) by the unconscious attitude he bore toward his father to whom he attributed motives present in his own mind. Qualities present in people recalling to the patient those he saw in his own father determined the attitude of the patient toward these people. This state of affairs extended over many years and the emotional state was of similar duration. Applying to this what Dr. Kempf taught, this patient's autonomic system would seem to be "conditioned" by attributes he saw in people suggesting unconsciously those possessed by his father, causing a change in the normal muscle tonus sufficient to bring about muscular discharge, that is, striking of palm with fist. This change of tension was the "feeling," in the present instance, of anger felt by the patient as a disagreeable sensation, the origin of which was unconscious to him. This gave a physiologic explanation for that which Freud empirically designated as an "unconscious wish." To him it was a psychologic phenomenon, and it was

gratifying to substantiate his clinical findings by means of physiologic data. In the case just cited, the more or less unconscious hatred for his father, his pathologically motivated ambition, his rebellion to authority, all were accompanied by an unconscious wish to remove a rival, that is, the irritating stimulus. This was the unconscious wish.

DR. FOSTER KENNEDY thought that it might be of interest to some of the members of the Neurological Society in this connection to hear of two soldiers who were under his care last year and who exhibited in a very astonishing manner a cooperation of endocrinologic disturbance and emotional disturbance. These men were admitted to the hospital with complete emotional collapse after having been blown up without physical injury. They were in identical conditions, almost mute, almost inaccessible; it was difficult to make them eat and emotionally they were given over entirely to the phenomenon of fear. In both cases the hair was standing erect on their heads and remained so for nine days. The hair of both these men was long and stood on end like that of a Zulu. After a few days rest in bed they recovered their usual mental and emotional balance and were able to state in each instance that their hair previously was normally tractable and flat.

DR. BERNARD GLUBCK thought that every one should become familiar with Dr. Kempf's monograph on the "Autonomic System in Its Relation to Personality" because of the very helpful synthesis he had accomplished of the various physiologic and psychologic researches into human conduct. He had occasion recently in teaching psychopathology to his students at the School for Social Service to use this monograph very effectively, and found it very helpful in approaching this subject from the physiologic standpoint as outlined therein. It made very much more acceptable to the average mind the central theme in the Freudian theory of conduct—namely, the wish. Dr. Kempf's discussion of the place for the craving of social esteem in human conduct was particularly illuminating, as it frequently was found to be the most prominent factor in problems of social maladjustments.

DR. S. ROTHENBERG thought that every one present was indebted to Dr. Kempf for his splendid presentation and he personally appreciated it, particularly because of having done some work in attempting to interpret the so-called mismating complex from a psychosexual point of view. This work was done at the National Desertion Bureau in New York. He found there were decided and peculiar condition reflexes in many of these people who could not adjust themselves to the marriage state, which would explain the underlying difficulties better than in any other way. Many interesting instances of that kind had been noted.

DR. KEMPF, in closing the discussion, expressed his gratitude for the kind appreciation given him. He thought that the hallucinations in Dr. Jelliffe's case could be interpreted as an auditory hallucination caused by the anal segment, which had become dissociated, trying to obtain the necessary stimulus for gratification. He had been forced to the conclusion that one could not get a psychosis from an organic or toxic cause in which the primary autonomic affective cravings had not been previously repressed. The study of paretics, deliria and arteriosclerotics showed that strong autonomic compulsions, which had been repressed or were perversely conditioned, had become dissociated and caused the hallucinations, etc.

Dr. Jelliffe's point about the localization of disease processes in repressed segments might be explained by the fact that an autonomic segment which

was more or less anemic or hyperemic, due to the vasoconstriction or vasodilation in this segment which existed because of its repression, disease, or over-tension, became a more fertile soil for bacteria.

As far as therapeutic principles were concerned, there were two schools; one believed in building up the health, confidence, self-control and integrity of the ego, and the other in getting a *transfer* from the ego so that it would no longer be afraid of allowing the repressed craving to cause awareness of its efforts. With the transfer, the patient became conscious of what his personality craved and by learning to analyze and know himself, he became able to make much more comfortable practical adjustments, without becoming fascinated by bizarre, asocial, or perverse stimuli.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, March 20, 1919

HUGH T. PATRICK, M.D., *President*

EXPERIENCES WITH THE AMERICAN RED CROSS IN ITALY.

DR. RALPH C. HAMILL addressed the Society on this subject.

TWO OPERATED CASES OF EXTRADURAL TUMOR COMPRESSING THORACIC PORTION OF CORD. Presented by DRs. CARL B. DAVIS and PETER BASSOE.

CASE 1.—Summary.—Paraplegia of two years' standing; loss of all sensation below the level of the fifth thoracic segment; visible and palpable subcutaneous tumor corresponding level of back. Operation: Removal of hour-glass shaped lymphosarcoma, partly intraspinal, connected with external tumor through two intervertebral foramina.

History.—A woman, aged 49, was seen by Dr. Bassoe on Jan. 24, 1919. In January, 1916, she began to complain of pain in the scapular region radiating around to the chest. The pain gradually grew worse. Unsteadiness of gait was noticed in June, 1916. Later the legs grew weak and after February, 1917, she could walk only with crutches, and early in 1918 became bedridden. Numbness in the toes began in January, 1918, and ascended. Control of the sphincters was lost at the time she became bedridden. A soft tumor of the back was accidentally discovered in June, 1918, located between the scapulae at the point where the pain was first noticed.

Examination.—Total paralysis of lower extremities and of abdominal and lower thoracic muscles. Marked knee and ankle clonus and positive Babinski sign. Legs extremely rigid and involuntary movements are caused by the lightest touch. Upper extremities and cranial nerves normal. Loss of all sensation below the level of fifth rib. A palpable tumor in back muscles, 1 cm. to the left of the fourth and fifth thoracic spines. Spinal fluid under low pressure, clear; cell count, 10; Nonne-Apelt globulin test, weakly positive; Lange test, weakly positive (0112211000). Examination of viscera negative. No glands palpable. Rectal and vaginal examination negative, except for a slight rectocele. Appearance of patient suggestive of anemia, but blood examination showed hemoglobin, 81 per cent.; red blood cells, 4,540,000; white blood cells, 9,100; blood pressure, systolic, 120, diastolic, 80. Roentgen-ray examination of the spine negative.

Operation.—A diagnosis of probable extramedullary tumor was made and the patient referred to Dr. Davis for operation, which was performed on Jan. 27, 1919. The tumor of the back was of a glistening appearance, measured 6 by 3.5 cm., and could be traced down to the intervertebral foramina above and below the fourth thoracic vertebra. After removal of the laminae of the upper five thoracic vertebrae the tumor tissue was found to be continuous with an extradural growth 4.5 cm. long, 1.7 cm. wide and 1 cm. in thickness, located posteriorly and to the left of the cord. It was readily peeled off, leaving the dura perfectly smooth.

Histologic Examination.—This was made of several intraspinal and extra-spinal parts of the tumor. On account of the unexpected appearance, sections were submitted to Prof. E. R. LeCount, who stated that the tissue was mainly lymphoid in character and that the tumor belonged in the class of lymphosarcoma—of the same histologic character as the kind described by Kundrath and later by MacCallum.

Subsequent History.—In spite of the surgical success of the operation the patient failed to show the hoped for improvement. Although she was kept on a water bed, very extensive bedsores developed. The rigidity of the legs diminished rapidly and after a few days the complete picture of a total transverse lesion existed, all of the reflexes being lost below the level of the lesion. The patient also developed a bronchopneumonia, and died on March 7, 1919. No necropsy was obtained, so it could not be determined whether any lymphomatous tumors existed elsewhere in the body.

CASE 2. — Summary.—*Paraplegia of one year's standing with prolonged remission; loss of all sensation below eighth thoracic vertebra; legs spastic; little pain; removal of extradural fibroma followed by slow but steady return of motion and rapid return of sensation.*

History.—A boy, aged 15, gave a history of having developed an almost complete paralysis of both legs during the summer of 1917. At the time there was some pain in the lower chest on both sides, but no sphincter disturbance. The paralysis lasted only a month. In the spring of 1918 the symptoms gradually returned and by the beginning of October both legs became paralyzed and spastic. Only slight pain was complained of.

Examination.—Made on admittance in November, 1918: Spastic paralysis of both legs with great rigidity whenever limbs are handled. The upper thoracic muscles contract. Beevor sign present. Bilateral Babinski sign, knee and ankle clonus. Abdominal and cremasteric reflexes absent. Loss of touch and pain sense total below costal margin and relative below level of sixth rib. Roentgen-ray examination of the spine negative. The spinal fluid gave a negative Wassermann test, cell count 7, weakly positive Nonne-Apelt and Lange test (0112110000). A brief course of antisyphilitic treatment was given without any benefit. A tentative diagnosis of spinal cord tumor was made and the patient referred to Dr. Davis for laminectomy, which was made on Dec. 19, 1918. An extradural tumor, of the size of the end of the thumb, at the level of the seventh thoracic vertebra was readily removed. The tumor was found to contain a cavity partly filled with clotted blood.

Histologic Examination.—This proved the tumor to be a fibroma.

Subsequent History.—The patient soon regained sensation and after three weeks slight return of voluntary motion in the left toes was noted. From that time on return of power has been fairly rapid, but the patient is still unable

to walk and the legs remain quite spastic. It was thought that the hemorrhagic cavity in the interior of the tumor might explain the two separate attacks of paraplegia.

DISCUSSION

DR. PETER BASSOE reported that the two patients operated on for extradural fibroma compressing the cord, shown to the Society by him in December, 1914 (*J. Nerv. & Ment. Dis.* 42:736, 1915), were well. In case 1 there had been no recurrence. Case 2, the patient with the large hour-glass tumor partly outside the spine, developed weakness in the legs four years after the operation and in January, 1918, Dr. Dean D. Lewis again operated, removing all visible portions of tumor. The patient again made a perfect recovery.

DR. H. N. MOYER cited a case operated on fifteen years ago by Dr. Bevan, which proved to be an exostosis at nearly the same level, with practically the same symptoms. It was about the size of one's thumb and compressed the cord into the form of a ribbon. The symptoms had been gradual in their onset, there being three or four years from the first weakness of the legs until the final, complete paraplegia set in. The tumor was chiselled away and the spinal canal restored. He had seen the patient a few weeks ago and there had been no recurrence of the growth. She still walked with a distinctly spastic gait, with a tendency to tilt a little on her toes. There was no incoordination or disturbance of feeling by usual clinical tests. The patient said improvement after leaving the hospital, continued for a little more than a year and a half, when she reached about the present stage. There were no fatigue symptoms whatever on walking and she had married and become a mother.

THREE CASES OF GLIOMATOUS CYST OF CEREBELLUM, WITH PRESENTATION OF SPECIMENS. Presented by DR. GEORGE B. HASSIN.

CASE 1.—History.—A gliomatous cyst of the cerebellum is a variety of so-called cerebellar cysts. The latter are roughly classified by the English neurologist—Williamson—as cystic tumors and so-called serous cysts. In the former a cyst is always associated with a tumor, while in a serous cyst no vestiges of a tumor growth can be found. The first of my cases concerns a boy, aged 17, who entered Cook County Hospital on April 14, 1917, complaining of headache, vomiting and vertigo. The headache had started six weeks previously, without any apparent cause, was confined to the occipital region and was continuous. Vomiting had persisted for three days, about from ten to twelve times a day, always associated with dizziness.

Examination.—This showed a well nourished boy lying on the left side with the head and legs bent. Any other posture would induce vertigo and vomiting. The mentality, reflexes, general sensibility, abdominal organs, the genitals and the chest organs were normal. The *right* side of the face, lower half, was slightly paralyzed, the tongue deviated to the right; ocular movements and pupils were normal. The right disk was slightly blurred; the left, normal. Roentgen-ray examination of the skull, examination of urine, and serums showed nothing abnormal. The patient died about five weeks after he entered the hospital, the entire duration of the symptoms having been about three months.

Necropsy.—This revealed in the left cerebellar hemisphere, a small glioma, the size of a bean, very vascular and hemorrhagic, surrounded by a cyst. The latter contained about an ounce of sterile hemorrhagic fluid. The larger por-

tion of the cerebellar lobe was destroyed, the vermis pushed to the right and appeared compressed. The right cerebellar lobe was normal. The cerebral tissue showed no changes. There was no hydrocephalus.

The postmortem findings demonstrate that the symptoms could not possibly have been caused by the glioma, as he took sick suddenly and the entire duration of his illness was only about three months. Evidently he had long harbored a symptomless glioma until a hemorrhage into the growth with subsequent cyst formation caused the severe subjective complaints.

Forced position in cerebellar lesions (in this case on the left) has been frequently described (Jackson, Gordon, Smith and others), being evidently due to the release from pressure of the fourth ventricle and of the opposite healthy side. The third interesting feature was the cranial nerve involvement (the seventh and the twelfth) which was also present in the other two cases.

CASE 2.—History.—Joseph D., aged 21, came under my care on July 17, 1918, complaining of continuous frontal headache of five months' duration, dizziness and vomiting. There was, besides, severe pain in the neck, total blindness (for two months), inability to walk and a history of two convulsions with unconsciousness.

Examination.—The patient was found in a fixed position, on the right side, semiflexed. The neck was rigid and painful on movement; the right sixth nerve and all the muscles of both the third nerves, except the levator palpebrae superioris, were paralyzed. The patellar and Achilles reflexes on the right were lost, pupils rigid to light and accommodation, pulse from 96 to 160. The fundi showed marked bilateral papillo edema. The mental state, sensation, speech and urine were normal. There was no ataxia, no tremor. Walking was impossible, partly from great weakness, partly from blindness. The patient died a month later in convulsions after a decompression operation. The necropsy showed a glioma in the left cerebellar hemisphere and a cyst involving the vermis and a portion of the opposite hemisphere. There was no communication of the cyst with the fourth ventricle, no hydrocephalus, or other changes in the brain tissue proper. The point of interest in this case, aside from the fixed position, was involvement of the ocular nerves, the third on both sides including the intrinsic muscles, and the sixth on the right.

CASE 3.—History.—Charles L., aged 34, a switchman, entered the nervous service on July 27, 1918, complaining of headache (one year's duration), dizziness, failing vision, salivation (one month), gait troubles and difficulty in talking.

Examination.—No abnormal postures. There were slight ptosis, protrusion of the eyeballs, bilateral papillo-edema, slight ataxia in the upper extremities, slight nystagmus, swaying gait, marked Romberg and dysarthria. The tendon reflexes were quite lively on the left side, the skin reflexes, except the cremasteric, were normal. The patient had several convulsions during the period of his illness. The serologic findings, heart, lungs and urine were negative.

Postmortem Examination.—Glioma and cyst of the lower half of the left cerebellar lobe, slight hydrocephalus in the left hemisphere.

COMMENT

The three cases exhibited similar lesions and clinically they were somewhat alike. In all of them, there was absence of the majority of the phenomena described in experiments on animals and by Babinski in cerebellar lesions in

man. Thus, there was no nystagmus (except slight in case 3), no asynergic or "demesured" movements, no adiadokokinesis, and even ataxia was not present in all cases. The regular manifestations were the subjective complaints (headache, dizziness, vomiting), papillo-edema, and the involvement of some of the cranial nerves. Neither the papillo-edema, nor the cranial nerve involvement has anything to do with the cerebellum; namely, they are not the result of the disturbed cerebellar function. The occurrence of the lesion of various cranial nerves in intracerebellar affections has been repeatedly reported in neurologic literature (Tooth, Patrick, Homburger, Rout, Kitchen and others), being generally looked on as the result of the direct or indirect pressure (Fernwirkung). For many cases such an interpretation of the cranial nerve paralysis may be true, but some cases could be more satisfactorily explained and understood in the light of experimental work on the cerebellum done by I. L. Meyers of this city and a member of this society, and also probably by Weed (Baltimore). As it can be gathered from their experiments, especially those of Meyers, a removed cerebellar lobe produces hyper- or hypoactivity of the corresponding cerebral hemisphere with the disturbances of neuro-muscular action as the result of the changed function of the brain. The regularity with which the cranial nerves are involved in cystic lesions of the cerebellum would strongly speak in favor of the conclusions arrived at by Meyers, from his remarkable experiments. The fact that in some cases of cystic or other cerebellar lesions signs of cranial nerve lesion may be absent is most probably due to the so-called compensatory function of the brain. Luciani states that animals even with extensive injuries to the cerebellum do not show, in the course of time, any clinical symptoms or signs whatsoever. Such animals will jump, and run, and play as if no damage had been done to the cerebellum. Babinski himself, points out the fact that a cerebellar lesion is sometimes accidentally discovered on the postmortem table without having been manifested clinically. Such compensatory functions of the brain must necessarily occur in older cases in which no cranial nerve involvement may be found. Recent cases, however, like the ones under discussion and as it can be gathered from the literature show this phenomenon with remarkable frequency and regularity.

DISCUSSION

DR. I. LEON MEYERS thought while a few of the symptoms in Dr. Hassin's case could be explained in accordance with his ideas of the cerebellar functions, others must be looked on as indirect symptoms. Regarding the deep reflexes, if we assume that the cerebellum exerts an inhibitory effect on the higher cerebral centers, the cause of their diminution or loss is of course obvious.

In the first case the patient was lying on the left side. This is a minor form of the rolling movements to the left, observed in animals and must be explained, in his experience, as indirect symptoms. He had pointed out some time ago that forced movements were not cerebellar symptoms, regardless of the numerous statements to the contrary. He had removed in a number of animals practically the whole lateral lobe of the cerebellum and they did not show any forced movements. They are constant, however, after lesions of the vestibular system. The animal after such a lesion, being more conscious of one direction than of the other, will of necessity turn its head and look in that direction and we have conjugate deviation of the head and eyes to the right or to the left as the case might be. In the comparatively large number of animals in which he observed rolling movements, conjugate devia-

tion of the head and eyes and allied phenomena, there was invariably a lesion of the vestibular nerve, the vestibular nuclei or posterior longitudinal bundle.

He had not made microscopic studies of the lesions. He could observe, however, that in small lesions the animal fell to one side, or turned its head to one side whereas in large lesions they turned around. He was convinced that the position in the case was due to some pressure on the left side. He thought the diminution of the reflexes was best explained by the theory that the cerebellum functionates as a checking organ on the motor centers of the cerebrum.

DR. PETER BASSOE asked if the cranial nerve symptoms were not very frequently due to hydrocephalus. In many cases of cerebellar tumor there had been so much hydrocephalus that it was easy to explain the involvement of the cranial nerve by the stretching of the nerves from the downward pressure.

DR. HUGH T. PATRICK was of the opinion that cranial nerve involvement in tumors and cysts of the cerebellum was to be accounted for on mechanical grounds.

DR. GEORGE B. HASSIN, in closing, said he wished to point out the regularity with which the cranial nerves were involved in cysts of the cerebellum, especially in cases not advanced. He did not agree with the explanation given by Dr. Meyers regarding the abnormal position of the body. The forced position of a patient with a cerebellar cyst was not the result of a disturbed cerebellar function, being rather a matter of comfort for the patient. He feels less distressed when lying on the diseased side, as a position on the healthy side causes increased pressure on the aqueduct of sylvius and greater distress.

The hydrocephalus could not be considered the cause of the multiple nerve involvement, because out of the three cases demonstrated only one showed a hydrocephalus which was unilateral and very slight. On the other hand, a regular cranial nerve involvement did not occur in hydrocephalus associated with other brain lesions.

As cerebellar cysts usually bring about an extensive and even complete destruction of whole lobes of the cerebellum, they could be utilized for the purpose of studying the clinical symptoms resulting from such a destruction. Solid tumors were less adapted, as they usually leave a larger portion of the cerebellar tissue comparatively intact.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, March 20, 1919

GEORGE A. WATERMAN, M.D., *President*

A NOTE ON DR. SOUTHARD'S ORDER OF EXCLUSION IN DIAGNOSIS. Presented by DR. LAWSON G. LOWREY.

DR. LOWREY stated that in mental diseases there is no definite system of presenting symptoms, but in considering any one case it is possible to narrow the possible diagnoses down to two or three groups. Dr. Southard has presented a scheme of diagnoses that contains eleven groups. Another possible order of grouping is one based on statistical frequency. This method would

result in different orders in different institutions, and is therefore objectionable. A better basis is one founded on etiologic factors.

Dr. Lowrey agreed with Dr. Southard as to the first four groups: the syphilopsychoses, the hypophrenoses, the epileptoses, and the pharmacopsychoses. In the last five groups, he also agreed: the geriopsychoses, the schizophrenoses, the cyclothymoses, the psychoneuroses, and the psychopathoses. As a fifth group, Dr. Southard has the encephalopsychoses, and as a sixth group the somatopsychoses. Dr. Lowrey thought that a reversal of this order is clearer, more logical, and simpler for teaching. He therefore suggested that the fifth group should be the somatopsychoses, and the sixth group the encephalopsychoses.

DISCUSSION

DR. ELMER E. SOUTHARD stated that the grouping that he had made was not based on a teaching consideration, but on reliability of tests of differentiation. The first four groups are social groups, the fifth concerns the neurologists, the sixth the problems of internal medicine. Such a grouping has the disadvantage of bringing into juxtaposition certain cases that might better be more widely separated. He had considered the order that Dr. Lowrey suggested and recognized the arguments for the transposition. The exact sequence of the grouping he thought less important than the acceptance of a definite classification.

A CASE OF ENCEPHALITIS LETHARGICA. Presented by DR. CHARLES A. McDONALD.

A case reported in brief and diagnosed as encephalitis lethargica, showing signs characteristic of this disease as described by Von Economo, Netter and Smith. The patient had cranial nerve involvement as shown by ptosis and diplopia. The lethargy was the initial and presenting symptom—mild at first—increasing in severity until she was comatose before death. Until the last two days of her illness she could be roused when she would be found to be oriented. In addition she had absent deeper reflexes. No Babinski sign. No bladder disturbance. White blood cell count a little less than 10,000 and spinal fluid within variation. Necropsy showed a general increased vascularity with no evidence of meningitis.

THE QUESTIONS OF DURATION OF ATTACK AND RECURRENCE IN MANIC-DEPRESSIVE INSANITY. Presented by DR. JOHN B. MACDONALD.

The problems of prognosis, duration of phase, length of interval, and recurrence in manic-depressive insanity were considered; also the light thrown on these problems by a study of 450 cases of this disease at the Danvers State Hospital. As to age periods, thirty-six males and eighty females, totaling 116, or 25.7 per cent., were 25 years or younger in age. Forty-four males and 132 females, totaling 176, or 39.1 per cent., were mature, namely, 25 to 40 years old. Forty-seven males and 100 females, totaling 147, or 32.5 per cent., were climacteric, that is, 40 to 60 years old. And four males, and eight females, totaling twelve, or 2.3 per cent., were postclimacteric, that is, 60 or more years old. Depression was the most frequent initial phase, being nearly twice as

common as manic or mixed types. When the initial attack was depression the second attack recurred with men in about 10.6 years, and with women in 10.9 years, but with an initial manic or mixed attack the average interval of recurrence in men was 6.1 years and in women 6.5 years.

On the other hand, the average duration of attack was considerably longer in the depressed cases. Only 62 per cent. of the depressed cases discharged recovered in six months, while 79 per cent. of the manic cases were so discharged. The recovery curve for manics reached its height between the fifth and sixth month; for depressions, between the seventh and eighth, when the initial attack occurred below the age of 40.

Beyond the age of 40 permanent recoveries were rare from first attacks.

Apparently it is most difficult for the manic-depressive case occurring after 40 to realize that he has passed the zenith of his powers, and that his plane of accomplishment is on a lower level.

In 295 of the 450 cases recurrences occurred. In 100 cases all the attacks were of the depressed type; in 128 all were manic.

To gain an idea of the tendency toward establishment of the complete manic-depressive cycle, a review was made of 100 cases in which the completed life history was known. Of these, seventy-eight showed in one or more attacks a variation of type, namely, a completion of cycle; twenty-two presented attacks of one general type throughout, but differing in duration and intensity. Of those showing uniformity of phase in all attacks almost two thirds were depressions.

In the case of depression, the extremes of duration of free intervals after first attacks varied from forty-four to two years for females and twenty-eight to two years for males. Following initial manic attacks, the extremes for females were twenty-eight and two years, and for males thirty-nine and one years.

Between the extremes mentioned, individual cases presented a bewildering variation of interval durations. Given certain antecedents, certain consequences do not uniformly follow in manic-depressive disease.

DISCUSSION

DR. H. R. STEDMAN asked the reader as to how many cases of recurrent paranoid conditions he had seen. He also asked regarding seasonal variations, and the possible influence of the season as a factor governing the recurrence.

DR. E. B. LANE stated that he had seen cases that completed a double cycle in a year. Environment played little part in the production of the condition. He thought that some somatic factor played a decisive part.

DR. E. E. SOUTHARD asked whether the analysis shed light on the problem as to whether manic-depressive insanity is a psychosis or a group of psychoses. Is Kraepelin to be credited for a new disease or merely a synthesis of disease? Would a study of heredity help?

DR. MACDONALD (closing the discussion) said that many of the cases did not seem to be distinct entities. He thought that the condition was probably a group of conditions. Completion of cycle was shown in seventy-eight out of 100 cases.

PHILADELPHIA NEUROLOGICAL SOCIETY

*Regular Meeting, March 28, 1919*J. HENDRIE LLOYD, *President***A CASE OF HEMIHYPERTROPHY.** Presented by DR. J. W. McCONNELL.

D., 7 months old, was born prematurely, but except for the fact that she was a seven months' child, was apparently normal. She was breast-fed and seemed in no way to be out of the usual, until at 2 months her mother noticed some difference in her lower extremities, this difference consisting of an unusual fatness of one leg as compared with the other. This condition has continued and has somewhat increased up to the present state.

The family history shows nothing important as to the father. The mother has had four pregnancies altogether, two of which were abortions; another was a child who is now 4 years of age, and the fourth is the patient of whom we are speaking. We found on examination a very marked asymmetry, our opinion being that the left side of the body was the pathologic one, this opinion being based on the appearance on the external surface of the leg of an unusual development which simulated a mass, the right leg not showing anything at all like it. The whole lower extremity, the buttock, the left labium, the left arm and slightly the left face, were quite larger than the corresponding members of the right side. There was little or no asymmetry of hands or feet.

This enlargement was smooth, firm to the feel yet not hard, was in no way boggy or edematous. There was no change in the color of the skin. The superficial blood vessels were not in any way dilated, and the surface temperature was the same as that of the right side. There was no loss of power and, so far as one can judge in a 7 months' old child, no incoordination. The tendon reflexes were present and apparently normal. The child was quite as intelligent as one would expect for the age, and otherwise than as indicated was perfectly normal.

The other living child in the family was between 3½ and 4 years old, was very large, weighing 51 pounds; the lower extremities were symmetrical, very well developed to the point of the suspicion of hypertrophy. There was no paralysis, but the knee jerks and Achilles jerks were not obtained.

The enlargement from roentgen-ray studies seemed to be entirely confined to the soft parts, as can be seen from examination of the roentgen-ray plate; the bones on both sides of the body were the same size—longitudinally and transversely—and the shadows of the soft tissues were no denser on one side than on the other.

TRANSVERSE MYELITIS IN A CHILD WITH THE ABSENCE OF THE TOE-PHENOMENON BY BABINSKI'S METHOD BUT PRESENCE BY OTHER METHODS. Presented by DR. ALFRED GORDON,

Boy, aged 9, presents evident signs of rickets: Scoliosis, one scapula larger and thicker than the other; curved tibia with convexity forward on both sides, more pronounced on the right. Prior to December, 1918, he was apparently in good health. At that time he complained of stiffness of the neck, became languid and apathetic. Rapidly he developed a weakness in the legs and had difficulty in walking. At the same time difficulty of micturition made its appearance.

At present the paralysis is not complete, as the patient is able to stand up when supported and to move his limbs when in bed, more on the left than on the right side. The knee jerks are increased on both sides. Ankle clonus is present and very marked on both sides. Babinski is absent on both sides, but the paradoxical reflex is present and distinct on both sides. Oppenheim's reflex was also distinct on both sides, but presently is obtainable only at times. Shäfer's sign is persistently present. Sensations to touch and pain, also temperature, are diminished from the toes up to the knees. The cremasteric reflex is much diminished on the left side. The sphincter of the bladder is disturbed: from retention the condition changed to imperative micturition.

The literature abounds in examples of this kind. There are cases in which irritation of the plantar surface produces extension of the great toe, but pinching the thigh and abdomen produces flexion of the same; cases of paraplegia, such as reported by Almeida and Espozel, in which Babinski's sign is positive, but negative after Esmarch's band had been applied and then removed; cases in which at first there was flexion and immediately afterward extension of the big toe. This phenomenon was observed in cases of cerebrospinal meningitis, of Brown-Séquard's paralysis, in cases of a lesion at the level of the seventh cervical vertebra. Guillain and Barré observed flexion in the ventral position and extension in the dorsal position of the patient. Landau of Switzerland, in a recent contribution (*Presse médicale*, 1917), reports a large series of cases in which the above-mentioned variations of response in the great toe or all the toes have been evident. He concludes with the following remark: "The procedure of producing extension of the great toe is not single; not only it varies from one individual to another, but it may happen that while all methods may be positive in one individual, only one may be present in another. In one case Gordon's method may give extension, while all other methods may be negative, etc. One cannot say that the toe-phenomenon is absent unless all methods have been used."

A CASE OF DISSEMINATED SCLEROSIS (?) WITH ASTEREOGNOSIS. Presented by DR. ALFRED GORDON.

A man, aged 23, laborer by occupation, commenced to feel various parasthetic disturbances, also developed a tremor in his fore limbs. The tremor disappeared at the end of several weeks, but the paresthesia remained. Six months ago he commenced to suffer from headache and diminution of vision, also from pain in the legs.

Examination reveals a fairly well-advanced secondary optic atrophy in the right eye (the left eye was enucleated in childhood). Pupil is normal. Visual acuity 8/200. The visual field is not contracted (Dr. LeFever). The gait is somewhat spastic. Ankle clonus on the left. Babinski on both sides. There is occasional incontinence of urine at night. Sensations: Superficial sensibility (touch, pain and temperature) is intact. The deep sensibilities are intact with exception of compass sense and stereognostic sense: at a distance of about 2 inches the two points of the compass begin to be distinguished as separated points; below that space, as one single point. Astereognosis is distinct and marked, especially for small objects, in the left hand, and sometimes there is difficulty of recognizing objects placed in the right hand. The senses of position and pressure are recognized by the patient. It seems, therefore, that stereognosis is not dependent on superficial and other deep sensibilities, but it is a sense apart. Moreover, the patient is fairly well able to recognize the form and consistence of objects, but is unable to name them. Asymbolia, therefore,

must be dissociated from astereognosis, which deals only with form and consistency of objects. The patient gave no history of venereal infection and the Wassermann reaction is negative.

As to the diagnosis, in spite of the absence of nystagmus and staccato speech, multiple sclerosis forces itself. We probably deal here with disseminated foci of sclerotic nature in various portions of the central nervous system. The optic atrophy, the astereognosis (pointing to the parietal lobe), the spastic paraplegia—all indicate the probability of the disseminated sclerotic processes.

CEREBRAL SYPHILIS WITH BILATERAL SEVENTH NERVE PALSY
AND INVOLVEMENT OF THE EIGHTH NERVES. Presented by
DR. J. HENDRIE LLOYD.

Dr. Lloyd's interest in syphilis of the nervous system has led him for some time to look particularly for involvement of the eighth nerves and apparatus of hearing, because syphilis of the auditory nerve and labyrinth is not so uncommon as once supposed. At a former meeting of this society he reported a case of almost total deafness coming on very acutely a few months after the primary sore, and due doubtless to a syphilitic lesion of the auditory nerves.

Dr. Lloyd presented two other cases, the auditory tests in which were demonstrated by Dr. Lewis Fisher.

CASE 1.—This case is particularly interesting, because the patient presents also a bilateral seventh nerve palsy, which is in itself a rarity. This patient, a colored man, aged 23, had a primary sore in September, 1918, and was admitted in October to the venereal wards of the Philadelphia General Hospital. He was given two doses of arsenobenzol, and left the hospital on December 1 against advice. There was no record at this time of any involvement of his nervous system. About three weeks later (December 24) he began to have severe and persistent headache, accompanied with vertigo and tinnitus in both ears. He staggered slightly, but at first there was no paralysis of the facial nerves. He was readmitted to the hospital about the first of the year, and at this time had well-marked paralysis of both facial nerves, more marked in the left. He had himself first noticed it from inability to hold a cigaret between his lips. The paralysis was of the peripheral type on both sides, involving the frontalis, the orbiculares, the upper and lower face and the lips. There has never been impairment of the third, fourth, fifth, sixth, tenth or twelfth nerves on either side. Taste and smell were not affected. There was no evidence of involvement of the cord, either in sensation or motion, but the knee jerks were unequal and the Achilles jerk on one side (the left) was totally abolished. The pupillary responses were normal: no optic neuritis. There was no dysuria. The laboratory reported on several specimens that the Wassermann tests for the blood and spinal fluid were positive, and globulin positive. At first the cell count was as high as 780—but later this had fallen to 36 per cubic mm. under treatment. He received six doses of arsenobenzol to date (March 21). Dr. Byrne reported a slight obstructive deafness. The positive but shortened Rinné with loss of intermediate notes, pointed strongly to involvement of the nervous mechanism. Dr. Lewis Fisher reported total absence of response to douching and turning, which would indicate a peripheral or eighth nerve lesion; but a definite presence of vertigo after turning, as well as preservation of a fair amount of hearing on both sides, shows that the nerves in their entirety were not involved; and he suggested a bilateral syphilitic lesion of the brain-stem on the mesial aspect of Deiter's nucleus, thus allowing for the

escape of the fibers for vertigo and most of the auditory fibers. The difficulty of excluding a purely peripheral lesion was the fact that both seventh nerves were implicated along with the eighth nerves. A syphilitic meningitis or neuritis, involving both the seventh and eighth nerves, would seem a probable lesion—and the fact that not all the cochlear-vestibular fibers were involved, but only some of them, would be in accord with what we know of the selective, or erratic, action of the spirochetes.

CASE 2.—Cerebral Syphilis with Eighth Nerve Involvement.—The second case was that of a Russian, aged 48, who was admitted to the venereal wards on Sept. 5, 1918, with a primary sore. He received mixed treatment and five doses of arsenobenzol. In December the sore was still unhealed, and there was enlargement of the lymph glands of the groin and neck. In March the patient began to complain of inability to walk straight. He staggered and fell to the left. Trouble with hearing had commenced earlier in the right ear, in which he had become quite deaf. Progressive deafness was later present in the left ear. There was loud tinnitus in the right ear, which kept him awake at night. The Wassermann test of the blood was reported negative, but the spinal fluid was positive + + + +. A curious phenomenon was complete abolition of one Achilles jerk (the left), just as in the other case. The knee jerks were free, especially the left. There is a low-grade double optic neuritis. The frontalis muscle on the left was smoothed out, but the orbicularis was not involved, nor the facial muscles, unless very slightly. The pupils reacted normally to light and on accommodation. All the other cranial nerves were normal. The pain, temperature and tactile senses were normal throughout. The gait was staggering, not ataxic, and there was a slight Romberg sign. Dr. Fisher found by testing that the right labyrinth was completely, the left partly, involved. There were no reactions to turning or douching. The case seemed to be a clear one of peripheral involvement, that is, of the labyrinths and eighth nerves in both branches.

It was a curious coincidence that both of these patients presented a unilateral absence of the Achilles jerk—without other marked evidence of involvement of the spinal cord. The explanation of this was not apparent, unless it meant slight beginning changes in the posterior columns or nerve-roots. But otherwise there was no evidence of tabes.

DISCUSSION

DR. LEWIS FISHER stated that he did not wish to be understood as disputing the diagnosis of Dr. Lloyd. He did, however, state to Dr. Lloyd in his report that from the ear standpoint he could not explain one of the cases presented on the basis of a peripheral lesion pure and simple. He demonstrated the reasons for his opinion when he examined the two cases in the turning-chair in the presence of the Society. Although the normal responses obtained from labyrinthine stimulation by turning were well known and accepted for a number of years, this war has offered an unusual opportunity for verifying them. An examination of some 60,000 prospective aviators in the Army has definitely shown that every normal individual had nystagmus of twenty-four seconds average duration after ten turns in a revolving chair. He simultaneously also experienced a vertigo—that is, a sensation of turning which is not in accord with fact—lasting between twenty-five and thirty seconds after ten rapid turns in a revolving chair. These phenomena are explained in the following way: When an individual was turned in a chair which was suddenly brought to a standstill, the lymph within the semicircular canals

of the labyrinth continued circulating by sheer force of momentum imparted to it by the turning. The moving lymph impinged against groups of ciliated hair-cells placed on little mounds at the ends of the semicircular canals. Just so long as this lymph continued moving, just that long there appeared the two responses noted—namely, nystagmus and vertigo. Both of these responses, therefore, originated within the labyrinth. The eye-movement impulse has the posterior longitudinal bundle for its destination. The vertigo impulse, which is a conscious sensation of motion, naturally, has the cerebral cortex for its destination. Each impulse travels very likely along a nerve-pathway of its own. All of these pathways are together within the eighth nerve. After the eighth nerve enters the brain-stem, where it breaks up into its constituent portions, these responses take different courses. Whenever there is a peripheral lesion within the labyrinth it usually affects *all* the ciliated hair-cells. It would be improbable for the pathologic process to destroy one set of hair-cells and leave the other set absolutely intact. The same is probably true of a lesion involving the eighth nerve itself. This uniform and proportionate involvement of all the responses is the distinctive feature of a peripheral lesion. When we say all the responses we also include the hearing. There have been observed, however, several cases of undoubted peripheral lesion where the hearing was not affected while the vestibular responses were practically absent. The association, however, of the auditory fibers with the vestibular fibers, although intimate, is not quite as intimate as of the two sets of vestibular fibers. This might explain the few exceptions.

In the case referred to (that of the colored man) we have the following findings. His hearing was quite good on both sides. When he was turned in a chair there was absolutely no nystagmus or eye-response, but there was present almost normal vertigo. The fact that vertigo was present indicated that the lymph was set in motion by the turning. How will it be explained then that this circulating lymph stimulates one set of ciliated hair-cells—namely, those for vertigo—and not the other? If the syphilitic toxin affected the labyrinthine contents themselves it would be unlikely for it to destroy the eye-movement fibers to utter extinction and leave the vertigo fibers practically intact. A more likely explanation of this phenomenon is that the pathologic process affected the eighth nerve distribution at some point where the eye-movement fibers have parted company from the vertigo fibers—namely, in the brain-stem. If our conception of the vestibular nerve-pathways be correct such a lesion would have to be bilateral in the region of Deiter's nucleus. This is purely an otologic suggestion to the neurologist and is offered only with the thought that it might fit in with the general neurologic picture, which this case presented. If the other general neurologic evidence points unmistakably to a peripheral lesion, probably the neurologist will help to explain these perplexing otologic data.

Another thought that was offered was this: Since it is well known that syphilis has a predilection for eighth-nerve tissue, could it not be possible that in this particular case the toxin had affected this eighth nerve distribution at more than one point—both peripherally and centrally? If this were true then everything could be easily explained.

The other case showed a marked impairment of *all* responses—hearing as well as nystagmus and vertigo. This was fully in conformity with the usual ear-findings in a case of peripheral lesion and the otologic examination simply corroborated the neurologic data in this case.

DR. GORDON asked Dr. Fisher whether he used the electrical current in examination of the vestibular apparatus; to which Dr. Fisher answered in the affirmative.

DR. GORDON asked him to tell the Society the comparative results between the two types of tests.

DR. FISHER replied that galvanic stimulation in vestibular cases was not generally employed for the reason that the electric current penetrates the tissues and stimulates, not only the hair-cells within the labyrinth, but also the nerve fibers within the eighth nerve and perhaps even the nuclei themselves within the brain-stem. In other words, it is an agency over which we have very little control so that the presence of a response on such stimulation does not indicate where the stimulus started. In some cases, however, it is very valuable, especially in those peripheral affections where there is a question whether the lesion is labyrinthine or retro-labyrinthine. In those cases it is the only means of differentiating between a lesion within the labyrinth itself and the eighth nerve.

DR. MILLS said he positively could not see why it could not be just at the junction of the fibers with the brain-stem. Omitting the Bárány tests the symptomatology seemed to be that of peripheral cases.

DR. FISHER responded that was the way it seemed to him.

DR. LLOYD stated he only wished to reiterate that he thought the simplest explanation of the first case was a syphilitic meningitis involving the seventh and eighth nerves on both sides where they emerged from the brain-stem. In support of this view it is known from other observers that syphilis of the eighth nerve is nearly always bilateral, and that it does not always involve all the fibers of the nerve, but sometimes only some of them. Thus the vestibular root alone may be involved, or in other cases the cochlear root alone. This selective action of the organism of syphilis is not unusual.

CEREBELLO-BULBAR POLIOENCEPHALITIS ORIGINATING DURING OR AFTER EPIDEMICS OF INFLUENZA AND OF POLIO-MYELITIS, INCLUDING THE RECORD OF A CASE OF EPIDEMIC ENCEPHALITIS OF THE LETHARGIC TYPE. Presented by DRs. CHARLES K. MILLS AND GEORGE WILSON.

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A CASE OF LETHARGIC ENCEPHALITIS. Presented by DR. P. N. BERGERON.

The patient was a single man, aged 34, formerly employed as a chauffeur, but lately engaged in automobile repair work. The family history may be of some interest; a brother died at the age of 19 years, after a short period of illness; his case was diagnosed "water on the brain" (this occurred about twenty years ago). The parents are both dead. The cause of death in either case cannot be now ascertained. There is a sister still living and in good health.

During the past eight or nine years, the patient suffered from a few attacks of rhinitis and pharyngitis. About two years ago Dr. Bergeron operated on him for lipoma of the cheek. Previous to last October the patient had had no serious illness. In October he fell a victim to the then prevalent "grippe," which disabled him for a period of ten days. This attack was not followed

by any serious complications, although he did complain of a slight pain in the loin; this pain gradually disappeared in a few weeks. After that he enjoyed good health.

March 8: The patient arose as usual, but soon discovered that his vision was so impaired that it was impossible for him to read. This, however, did not alarm him at the time. Two days later he complained *only* of this partial blindness.

Examination of the eyes showed that vision was 20/40 in both eyes. The eye ground presented a congested appearance at the nerve heads. His temperature was normal, and the pulse was 86. By close questioning it was brought out that he felt more dull than usual, but did not feel ill. Test for the reaction of the tendon reflexes showed no abnormalities. There was no paralysis, nor muscular weakness. The tongue was projected normally, and was covered with a heavy white coating.

March 13: The patient was unable to leave his bed. His condition grew steadily worse, and he was taken to St. Mary's Hospital. He lay on his back in bed, his legs fully extended, the arms flexed across his chest, his eyes closed and he was apparently sleeping. When asked, "How do you feel?" he would reply, "Good, but dull." He spoke slowly but intelligently, and immediately relapsed into an apparent sleep. He would remain in this condition until spoken to again. All the reflexes were normal, except the pupillary reflex, which was very much slower than on the previous day. When asked to open his eyes, voluntary effort on his part was fruitless. If the eyelids were lifted by the examiner and held so for a few moments, the patient would subsequently open them if spoken to, but soon closed the eyes again. The tongue was projected with difficulty, the lips were parted with marked effort on the part of the patient. When asked if he recognized the persons who were around his bed, he answered in the affirmative.

The temperature was 100 F., and the pulse was 86.

March 14: The condition of the patient remained unchanged. However, at this time it was noticed that the bowel and the bladder were incontinent. The patient was seemingly unaware of this. The sleeping was accompanied by snoring. The nurse observed that the patient had frequent attacks of tremor of the whole body that shook the bed on which he was lying. The temperature was 100 F., and the pulse was 86.

March 15: The condition of the patient remained much the same as on the previous day, except that he seemed more lethargic. It was also noticed, for the first time, that the arms and legs were rigid, and it required an effort on the part of the examiner to flex them. There was no voluntary movement of the eyes. The eyelids remained closed, unless lifted by the examiner. It was noticed that the right eye could not move toward the right and showed an apparent internal strabismus. Pupillary reaction was very sluggish and the pupils were irregular. The head moved easily in all directions, showing that apparently there was no rigidity in the muscles of the neck.

At this time the mental condition was very cloudy. If spoken to, his answers were unintelligible. When the spinal cord was tapped, he took no cognizance of it. Incontinency of the bowel and bladder still continued. The temperature was 101 F., and the pulse was 110.

March 16, 17 and 18: The condition of the patient during these days changed somewhat. He was not so sleepy as heretofore, he responded more quickly, and settled his business affairs, also questioned those who were around him. The rigidity of the arms and legs was more marked. The eyes opened

more easily. The weakness of the right eye had entirely disappeared. Vision did not grow worse. No rigidity of the muscles of the neck developed. The pupillary reflex seemed to quicken. There was no apparent difficulty with deglutition. The temperature gradually rose to 103 F., the pulse to 160, and the respiration to 60. On March 19, at 5:30 a. m., he died suddenly.

Urinalysis.—There was a trace of albumin, otherwise it was negative.

Blood Examination.—Red cells, 4,160,000; leukocytes, 12,400; hemoglobin, 85 per cent. Differential Count: Polynuclear leukocytes, 77 per cent.; large lymphocytes, 8 per cent.; small lymphocytes, 15 per cent. A culture made from the blood was negative. The Wassermann test of the blood was negative.

Spinal Fluid Test.—Fluid was clear, and it reduced Fehling's solution. Globulin was increased. Cell count was 13 cells per cubic millimeter. No bacteria were found. No growth found on culture. Wassermann test of the fluid was negative.

TWO CASES OF INTRACRANIAL INFLUENZA SEQUELAE. Presented by DR. S. F. GILPIN.

CASE 1.—G. C., a white man, aged 57, came to the clinic on March 12, 1919. He gave a history of always having good health until he had influenza during October, 1918. He was in the house two weeks, recovered and worked until January 3. During December he had sharp pains in the back of his head and neck and across the eyes. He was treated by an osteopath. During the night following the fifth treatment he felt queer in bed, arose and went down stairs. Here he became dizzy and could not walk. He was put to bed and vomited five times. He had double vision for one week after this. He complained of pain in the head and back of the neck and of paresthetic sensations well distributed over the body. He was unable to work.

Examination showed ataxia of both gait and station. Pupils were unequal and reacted slowly to light. No diplopia was seen. There was moderate ptosis of the right upper lid. According to Dr. Heed the central retinal vessels show the changes of arteriosclerosis. The patient had a tendency to choke when swallowing liquids. No palsy of muscles of throat could be determined by examination. Touching his left hand produced the sensation of heat to the patient. His sphincters were controlled. The blood and fluid Wassermann reactions were negative. The fluid showed 5 cells per cubic millimeter and an increase in globulin. Both smear and culture of the spinal fluid were negative.

CASE 2.—This patient, A. K., was a white man, aged 25. He had a history of good past health until he had influenza last October. From that time he complained of weakness and nervousness.

For the past five weeks he complained of a pulling sensation in the eyes with severe headaches. He had noises in his ears like the boom of a cannon, like music, and like the singing of birds.

His physical examination was entirely negative with the exception of a primary optic atrophy. The blood and fluid Wassermann reactions were negative. He showed no cells and no excess of globulin in fluid. There was no knowledge of optic atrophy in this patient until his eye symptoms caused him to go to the Wills Eye Hospital a few weeks ago.

The question was the relation of the attack of influenza to the present condition of the nervous system in these two patients.

DISCUSSION

DR. ALFRED GORDON asked how soon after the influenza infection the present disturbance developed.

DR. GILPIN replied that they had influenza in October and in the one case the man went back to work in January. That was two months after the infection the symptoms developed. The other patient had influenza at practically the same time.

DR. WILLIAM G. SPILLER said he had seen three cases of lethargic encephalitis. He thought the case Dr. Smith had shown from his service at the University Hospital at the last meeting was probably of this type. That boy had entirely recovered.

A man, aged 43, was referred to Dr. Spiller by Dr. Shumway. About February 15 of this year diplopia developed suddenly. The man remarked that he saw double on looking out of the window. He had bilateral ptosis and weakness of all the ocular muscles, especially of the superior recti muscles. At this time he had slight headache and great drowsiness, and he soon became delirious. He would drop off to sleep at any time and it was difficult to keep his attention. If he were spoken to he would answer correctly and then would lapse into this peculiar mental state. If left alone he would talk to himself in a rapid low voice. He would talk in this way much of the night. There was no facial palsy at that time.

He showed the characteristic progression of cranial nerve palsy. On February 22, he developed weakness of the right side of the tongue. On March 3, a beginning paralysis of the right facial nerve was noticed, and within a few days this weakness became complete paralysis, and by this time the ocular palsies had disappeared. He had 42 cells in the spinal fluid to the c.mm., 96 per cent. of which were lymphocytes. The laboratory findings were those of syphilis, but he probably had had a syphilitic infection for years, and his acute symptoms were those of lethargic encephalitis in a syphilitic man. His temperature was 101 F. when he came into the hospital. He was well on the way to recovery.

The third case was from West Virginia. A man, aged 38, on Feb. 14, 1919, developed suddenly headache and nausea; following this he became very drowsy, he had diplopia, the retina in each eye became congested, and he was delirious. The diplopia improved after four or five days. He had a temperature of 101 F. He then developed a complete left facial palsy. When he came to the University Hospital he had free lacrimation and conjunctivitis, more marked on the side of the facial palsy. There was no rigidity of the neck and there were no signs of meningitis. Paralysis of the left cervical sympathetic nerve was present from the lesion of the left side of the medulla oblongata. Dr. Spiller expressed the opinion that there can be no doubt of the existence of sympathetic fibers in the lateral portion of this part of the brain. The sympathetic palsy was on the side of the facial palsy, but the sympathetic palsy predominated, and the palpebral fissure on the left side was smaller than that on the right. The sympathetic palsy has now disappeared. Dr. Shumway found the internal recti muscles were weak. The cerebrospinal fluid gave 9 cells to the c.mm. The findings were negative for the Wassermann reaction.

This man had been in a neighborhood where influenza had been prevalent. Dr. Spiller expressed the opinion that lethargic encephalitis probably would be recognized as a form of influenza, one in which the organisms were carried

from the nasopharynx directly to the brain, without causing gastro-intestinal or pulmonary symptoms.

In neither case was there involvement of the fifth nerve, and Dr. Spiller expressed the opinion that possibly this nerve would be found to be less frequently involved than were nerves whose nuclei were nearer the fourth ventricle or aqueduct of Sylvius. The findings in Dr. Wilson's sections showed that the lesions were more intense near these cavities. They resembled greatly those of the encephalic form of poliomyelitis, from cases of which Dr. Spiller had examined microscopic sections, but he did not believe poliomyelitis and lethargic encephalitis were identical.

DR. CHARLES K. MILLS said that what he had to contribute to this discussion had already been given in the paper by Dr. Wilson and himself. One thing that impressed him was the comparison to be made between the so-called lethargic cases reported here by Drs. Spiller, Potts and Dercum, and others, and the bulbar and pontile symptoms reported in the cases of Drs. Mills and Wilson. Omitting the lethargy, the bulbar and pontile symptoms were much the same in both the lethargic and nonlethargic cases. In four out of the five cases were marked cranial nerve palsies—seventh, sixth, third and, in one instance, fifth. Most of the cranial nerve symptoms tended to recover, as in many of the lethargic cases. Dr. Mills said that he believed one point was most interesting, the very last point referred to by Dr. Dercum, namely, that in the cases which have been recorded in the lethargic disease, the symptoms have come on in part late. This was equally true of the cerebellobulbar and pontile cases described in the paper by Drs. Mills and Wilson. Certain symptoms appeared and then partially subsided or disappeared, but returned with increasing severity after a variable time.

DR. EDWARD A. SHUMWAY said that as Dr. Spiller had reported fully on his second case, he would confine his remarks to the first one. The man, when he first came to Dr. Shumway, showed paralysis of his right external rectus muscle. It was often difficult to determine the exact muscles involved in these patients because of the pronounced lethargy, which interfered with the determination of the double images, on which the ophthalmologist depends for his diagnosis. He subsequently developed disturbance of the oculomotor nerves, the left eye being most affected at first, especially in its upward movement; later, however, the right eye showed more limitation of its upward movement. There was partial ptosis of both eyelids, constant lateral nystagmus, increasing when the eyes were turned laterally or upward, and normal movements of the pupils to light. Ophthalmoscopic examination showed some blurring of both nerve edges, with hyperemia of the nerves. Within a few weeks, the oculomotor muscle palsies had improved very materially, as Dr. Spiller had said. Dr. Shumway alluded to a case, which he had seen in the Lankenau Hospital, which seemed to be of the same type, although he had understood that Dr. Potts, who had also seen this case, had thought at first it was a case of cerebrospinal syphilis. The patient came to the hospital with complete ptosis on both sides. There was marked disturbance of the rotations of the eyes, with involvement of both the oculomotor and abducens nerves, and the eye-grounds showed some hyperemia of the nerve heads. The patient was markedly stuporous, lying for hours at a time in this condition, although he would rouse sufficiently to respond if questioned sharply. He gradually improved apparently, the eye movements and lid movements were better, but suddenly the temperature shot up, one evening to 107 F., his breathing became affected, and he died. Unfortunately, no post mortem examination could be obtained. Another case

followed directly on an attack of influenza, in a young girl. In this case, however, there was a simple external rectus paralysis, without lethargy, and the paralysis was improving. Similarly, one other case had been seen at the University eye clinic, with paresis of both external recti, following influenza, also without decided mental disturbance. Ophthalmologists had been struck forcibly by the great number of muscle palsies, which they had seen recently, which seemed to be associated with the epidemic of influenza.

DR. FRANCIS X. DERCUM thought that the two cases presented by Dr. Gilpin were very properly referable to "grippe." Both presented organic changes suggesting encephalitis. Dr. Bergeron's very interesting case was another example. When Dr. Dercum saw this case the ptosis and inability of the patient to move the eyeballs freely and the sluggish reaction of the pupil to light were very striking. The man was very lethargic and somnolent, and ran a very moderate temperature; the whole picture at once suggested encephalitis lethargica. In a case which Dr. Dercum saw only a few days ago, a man, aged 42, and subsequently admitted to the Jefferson Hospital, a similar picture was present. There was a weakness of the eye muscles and lessened response of the pupils to light; and the mental condition again was one of somnolence and hebetude. At times this man was talkative at night, but soon dropped off and was again stuporous. A third case that Dr. Dercum had seen was still under observation at the Jefferson Hospital. Doubtless more of these cases will now be met with.

An especially interesting and important point was the extreme lateness of the development of the symptoms. Encephalitis lethargica does not seem to be a new and independent disease, but a sequel of "grippe." It would seem that in such cases the recovery which the patient makes from the initial attack is not a real recovery. Immunization has been incomplete, and later when the defensive reactions of the patient prove insufficient, the germs, again, gain the upper hand. The practical inference is that every patient who has passed through an attack of influenza, should remain in some degree under medical observation, should be full-fed, should be made the subject of a tonic and reconstructive treatment, and should not resume his occupation too soon. After encephalitis has occurred very little can be done for the patient. There is one point, however, which should be considered and that is whether drainage should be practiced. Dr. Dercum said that the drainage could do no harm and might do good; and he felt strongly inclined to urge it.

DR. CHARLES S. POTTS said that in reference to the case Dr. Shumway had mentioned as having been diagnosed cerebrospinal syphilis, he had seen the patient the day he was admitted to the Lankenau Hospital and suggested that it might be syphilis. After consideration Dr. Potts came to the conclusion that it was one of the cases of so-called lethargic encephalitis. The history of that man was of some interest, especially in view of what Dr. Spiller had said in reference to influenza. There seemed to be distinct relationship between the two as he had had influenza five weeks previously in one of the camps. He came home supposedly well and the first symptom of his attack was noted when he went to work, awoke one morning unable to open his eyes. He became stuporous and was sent to the Lankenau Hospital. He had no headache, no rigidity of the head and limbs and was intensely stuporous so that it was impossible to make a satisfactory examination of all the cranial nerves. Report on the condition of his eyes then stated that there was a third nerve palsy on each side, with ptosis, also paralysis of the fourth and sixth on one side, but that report said there was no evidence of optic neuritis. The cerebrospinal fluid was examined the next day and contained 76 cells—all lymphocytes—it

reduced Fehling's solution and was otherwise negative. He improved, became brighter mentally and the ocular palsy improved. On March 4 the fluid was again examined and was normal. The next day the patient became worse, the temperature rose to 107 F., the spinal fluid was examined again and contained 46 cells and in addition to the lymphocytes, contained a few polymorphonuclears, and it reduced Fehling's solution. He died shortly after that. Another symptom he had at the time Dr. Potts saw him was twitchings of the right side of the face. They could not make out any paralysis, but he had twitchings for several days.

DR. ALFRED GORDON said he had three cases which he wished to report. One case presented a temperature. The man in the very beginning had somnolence and then on the sixth day ptosis. There was also paralysis of the internal rectus of the left eye. The patient made a recovery in four weeks.

The second case was that of a woman, aged 27, who had a child with normal delivery; lochia normal. On the third day she developed a temperature of 104.1 F. and somnolence. She also had difficulty in swallowing and difficulty of speech because of awkwardness of the tongue. She presented involvement of the third, ninth and twelfth nerves.

The third case was a patient who showed temperature of 99.5 F., somnolence, palsy of the left external rectus, and difficulty in swallowing. In addition there was an anesthesia of the left cheek.

In all cases there was elevation of temperature. Whether it was "grippe" or not was difficult to determine, particularly in the case of the woman with the normal confinement, who suddenly developed high temperature. The three cases went into the category of lethargic encephalitis described particularly abroad. Encephalitis had been known for a long time. Accounts of its following infectious diseases can be found in the literature. Why should it be considered as a new disease? The disease has been known, but attention has not been particularly drawn to it. Netter wishes to consider it as an autonomous affection, but there is no substantial reason for it. In regard to the pathologic findings, Marie made exhaustive studies, and Dr. Gordon asked Dr. Wilson whether he also found lesions in the lobus niger.

DR. WILSON replied that no lesions were found in the lobus niger.

Book Review

SYMPTOMS AND THEIR INTERPRETATION. By JAMES MAKENZIE, M.D., LL.D. (Aber. and Edinb.). Ed. 3, New York, Paul B. Hoeber, 1918.

Centuries ago a wise man propounded the theory that "of making many books there is no end," and as years were added to years this idea like many others expressed by him was amply verified. He put no qualification on the books, however, as to whether they would be good or bad, and it is not unreasonable to suppose that on a time a publication was handed to our much-suffering friend Job, who in the double capacity of invalid and critic, gave utterance to the famous "My desire is that mine adversary had written a book."

Were Job living today he would have accepted "Symptoms" most gladly and would have recommended it to his friends as a volume worth careful perusal. It is indeed a pleasure in these days of all kinds of literature to find a scientific book that can be read at will that is sufficiently practical to be interesting and of sufficient interest to make it readable without tremendous concentration of mind and consequent brain tire. The size of the book is comfortable to the hand; the topography easy to the eye; the text readily understood; the few illustrations quite graphic but hardly as happy as the occasional clinical pictures with which the 300 pages are interspersed.

The especial value of the publication is to the practitioner of general medicine who, although in constant touch with the development of a disease process, oftentimes undervalues some of the most important features or perhaps misinterprets them. To him the earliest premise of the book will be the keynote of the whole matter—"An inquirer should keep his mind free from bias and ready to review his most cherished beliefs." To be sure, we all may not accept the author's hypothesis as to the purpose of reflex symptoms—he admits the necessity for correcting some of his own pet theories; we may not all believe with him that "the pain of uterine contraction is a distinctly referred one," or that "a persistent pain along the supposed course of a nerve with tenderness on pressure often attributed to neuritis is not infrequently not due to neuritis, but is the reflex phenomenon arising from some diseased viscus," yet his arguments in favor of viscerosensory and visceromotor reflexes are most convincing and his clinical proofs are beyond immediate questioning. In evaluating symptoms the author emphasizes the need for the elimination of the neurotic habit induced by prolonged suffering before calculating the real importance of reflex phenomena. In a way, this is not new, but is worth having attention called to it.

One could analyze the book from each of its various chapter headings with many commendations and little fault-finding. Suffice it to say, that all through the volume the writer's premises are well founded, and his arguments are interesting and frequently unique and his deductions most valuable.

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THE PHYSICIAN AND HUMAN CONSERVATION *

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PASADENA, CALIF.

On this occasion, when you might expect me to speak in the line of the specialty, the subject of human conservation may seem rather remote. It has none of the interest of novelty and has no problems for the laboratory except that oldest of laboratories in which the problems of life are being solved. The subject is familiar, but life is made of familiar things of which we find it necessary frequently to remind ourselves, and experience teaches that it is much more profitable to be reminded than to be instructed.

The existence of so much disease that is preventable, the increasing amount of insanity, feeble-mindedness and imbecility that are usually evidence of individual and family degeneracy, the poor showing of young men in our recent war conscription, and other conditions of bad import too numerous to be recounted have led me to consider at this time the subject of disease prevention and human conservation. This can only be a suggestion, as there is no time to frame an argument; it can only be a hint of the facts, not a presentation of them.

My subject is not so remote from our specialty as it may seem, for we are interested in disease and its prevention, and conservation deals with both. The great merit of prevention is that it begins at the source and that is the place to stop trouble. To bring a dying man back to life and health is more attractive and picturesque than the homely process of preventing him from being sick. Just a plain healthy man may be useful, but he is not necessarily interesting — society, however, prefers that a man should first be healthy. We have wasted a vast amount of human material in the past through mere neglect; we have wasted as much more through being satisfied to care for the wreckage, without considering how to stop the supply. The result has been that while we have been building hospitals and asylums, disease has been increasing with the growth of population, so that the age-old "pestilence that walketh in darkness," in the form of devitalizing con-

* Presidential address before the American Neurological Association at Atlantic City, N. J., June 17-18, 1919.

ditions, has been breeding disease and degeneracy and pouring its victims through the gates of charity.

There is much unused power in the medical profession in this country, some of which might surely be devoted to educating the public to understand the importance of human conservation. Resolutions of societies and well-meaning presidential addresses, unassisted by organized effort, will probably get no farther than to illustrate the infant death rate of good intentions.

The physician witnesses the growth and the unmaking of men in a way that others do not. He sees that simple, wholesome living produces healthy and effective lives. He sees untrained and therefore incompetent men and women struggling in a bad environment, becoming prematurely old, octogenarians at forty. He sees children who are underfed and dwarfed in mind and body repeating later in their own incompetence the incompetent lives of their parents.

We doctors have in the main been carrying on an ambulance service in the past, that is, we have been picking up the injured. It has been a great work unequaled by any other human agency. It has, however, been a partial service, in that it has dealt mainly with results and has achieved relatively little in the way of prevention. This was unavoidable as it has been only within the past few decades that our knowledge of the obscure sources of many diseases and degeneracies has permitted the formulation of a general plan for health conservation that would accomplish such results as are today possible.

PLAN FOR CONSERVATION OF HEALTH NEEDED

A conservation plan should be much more than one to prevent contagious diseases or even to prevent all disease. It should be a plan for human reconstruction in the broadest sense, not alone for cripples or for defectives, but for rebuilding the bodies of all the people, beginning with childhood and not necessarily ending with completed growth. Such a plan should be administered by the government. The business of the profession will be to secure the adoption of a plan. It will require the united efforts of physicians acting in every community to educate the people and show them what human conservation means. Unless the general public can be convinced of the necessity for it, the government will be slow to put a constructive program in force.

The large number of our young men who could not pass the examination for the army during the recent war furnished convincing evidence that there is in this country a great deal of poor physical development. It is, too, quite possible that some defects were overlooked, but certainly enough were exposed to show the physical vigor of Americans

is not what it might be or what it ought to be. The fact that so much degeneracy was shown to exist in persons of the ages of those examined, would indicate that there are others older and others younger who are equally at fault physically. The humiliating revelations of these army examinations are an admonition to the country to see that the boys and girls of our day have the training that will enable them to meet successfully the tests of active life.

There is fortunately a growing interest in the medical profession in preventive measures which is reflected in activities of various states and in that of the general government. The health work in cities, the community nursing, the follow-up work of dispensaries and hospitals, the new interest in housing one of the biggest of human problems, these and many other activities are educating the public for the comprehensive program that will some day come.

NATIONAL HEALTH PLAN

We have also in recent years been moving toward a national health plan in the activities of the marine hospital service, the work of the Surgeon-General's Office and the annual health lectures provided by the American Medical Association. The next step should be a comprehensive plan for the entire country. It is not possible nor necessary to suggest a scheme here, but if the profession could induce congress to appoint a medical commission to investigate the need for a health program, such a body could easily produce evidence of the necessity for it and secure action.

BEGINNING OF PREVENTIVE MEDICINE

There is no finer chapter in medical history nor one that is more significant than that which records the first achievements in prevention of disease. When the genius of a country doctor connected the milkmaid with the cow, and devised prevention by vaccination, he took the first step toward doubling the usefulness of our profession by showing that some diseases may be prevented. What Jenner demonstrated was not only a matter of science but a social fact of great importance. The prevention of disease earlier received an impetus from the old Venetians when they put the word quarantine in the dictionary. They might have waited until the plague had developed in the city and then have administered their remedies; they preferred prevention.

SOCIAL LOSS FROM DISEASE

Eleven thousand young men applied for the Naval Reserve in 1913 and only 316 were found physically fit. It has been said that these tests are severe. They are. Should the United States lower its

standard to fit our degenerate youth or should we raise the physical level of our manhood? These tests are probably not higher than those any normal young man should be able to pass.

The annual social loss from disease is equal to the destruction of a great war. This is all the more regrettable, because most of it is preventable. It is a serious thing that there are more than 500,000 deaths from preventable diseases in this country every year, 400 times the number that were lost on the Titanic, a tragedy that shocked the world. Three hundred thousand babies die before they are a year old, a loss of which the figures are a poor measure. More people die before the fiftieth year in the United States now than in the time of our grandfathers, though in several European countries more people live beyond the fiftieth year than was the case two generations ago.

Ripley says that of 100 Jewish babies born in Massachusetts, fifty will be alive at the end of seventy years. Of 100 babies other than Jews born in Massachusetts, fifty will be dead in forty-seven years. Why is there this difference of twenty-three years under essentially the same environment? Is it due to a more intelligent care of health and a better scheme of life?

There are said by school authorities to be 500,000 feeble-minded children in the public schools of this country, and it is probable that there are several times that number in the adult population. These morons breed true to the laws of degeneracy in families of ten and fifteen children. Where will this country find room for the 2,000,000 or 3,000,000 degenerates of this class that we will have two generations from this time? Has this Association ever called public attention to the danger that the feeble-minded may pollute the healthy blood of this nation?

Man seems to be the hardest of all animals to kill. Human beings will survive mistreatment that would kill pigs and cows. If a man kills another with an ax or a pistol, he is punished; if he should kill a man with a slum house, he is not punished, though the men who rent slum houses are so much more dangerous than men with axes and pistols are relatively harmless creatures. The owners of slum houses help to fill our hospitals with sick and insane, and in addition they furnish a large supply of degenerates who prey on society. A general plan of health conservation should remedy these and other like conditions that cause much of the disease we see in public hospitals and much of the degeneracy that we meet outside of them. These cases of disease are the natural drift from such sources as bad housing and its many attendant evils that are spreading like an epidemic not only in cities, but on farms and in villages. Such insanitary homes with their inadequate care of children are the causes of much stunted growth,

incompetence and disease. Here also only a comprehensive program of health conservation can correct these evils.

No discussion of this subject could properly omit mention of our educational system: While the government plan of health conservation could not be made to apply to education in the states, it could by suggestion and advice make health conservation in all the states the first consideration in education. Indirectly, therefore, a government plan would determine that the states did their duty.

HEALTH CONSERVATION SHOULD BEGIN IN PUBLIC SCHOOLS

If we are to prevent physical degeneracy, or rather if we are to stop that which is now going on, we must begin with the children in the public schools. Their health should not only be guarded, but the training of their bodies is quite as important as the training of their minds. This should appeal to the intelligence and public spirit and the essential loyalty of every physician, less, however, as a medical practitioner, than as a man who understands or ought to understand the importance of medical sociology. Every man and woman who walks the streets today is essentially a product, physically and mentally, of our schools. Has our educational system been a success—if not, why not?

The power and much of the genius of a nation depends on the health of its citizens, and the preparation for effective living and national achievement should be made chiefly during the growing period. These little folks who are the raw material of our future citizenship are naturally outdoor animals. Their mental activity is founded on physical activity. Our schools will never be organized on the proper basis until we recognize and put in practice the fundamental principle that physical training should be the foundation of mental training.

Every medical man should have an immediate interest in existing methods of education, and especially in the conditions under which those methods are applied, and of all physicians the specialist in nervous and mental diseases should have the greatest interest. Any one of the younger specialists of this Association could go into any public or private school today and select with an approximation to accuracy a pretty large group of pupils who will probably be his patients or his contemporaries' patients fifteen or twenty years from this time. He will need to interpret the significance of the nervous attitudes of the children, the instability of nerve centers shown in the facial expression and in the general irregular muscular movements and other familiar signs of imperfect development. One sees in these cases the results of half-starved nerve centers, and neglected muscular activities, struggling in vain for normal expression in a turmoil of wasteful effort.

Hundreds of thousands of young people are yearly going out into the world unprepared for life because their only training in school, if training it may be called, has been a system based on word symbols. There is little or no discipline for young people in the unrealities of books; they cannot connect them with life, for to them life is in doing. The failure that so many of them experience in after years is also due, in large degree, to a lack of proper physical development. From the bad mental training and the neglect of the body there results much retardation, mental stagnation and physical disability, which make incompetents of many and invalids of others, while a few, through the miracles that nature daily performs, get into the small class of successful people.

The commission appointed by the National Educational Association says in a recent report that it would focus secondary education on the great social objectives, and it places the health of the young at the head of the list. This is encouraging for our teachers as a class have been slow to recognize, if indeed they do recognize, the important fact that health and sanity and success in life depend more on good physical development than on all the book knowledge that has been or can be packed into the healthily rebellious minds of the young. It is true that manual training and kindred instructions is given in certain schools, but it has not reached the great mass of boys and girls of this country.

It is a fundamental fact in human development that young people have a positive hunger for doing things, for learning as the race learned, to *think by doing*. The greatest discovery man has made to date was the first tool he fashioned. It was the chief means of the early development of the race, and so deeply is this primitive talent planted in the brain that every psychiatrist knows that the ability to do things is one of the last faculties lost. In teaching the young how to use the hands, the teacher is leading them over the long way the race came; the teacher helps them to short-circuit the race process. In this way even a dull boy can be taught to do some one thing well, and such training will save many from the failure that has condemned thousands to insanity, crime or pauperism.

Any physician who has seen much of the insane in public institutions must have been impressed with the large number of this class whose mental disorder, where the basic cause could be explained, has been chiefly due to the fact that they had never learned to do any one thing well. The dullards and incompetents that come from our public schools would, if they could speak, tell a tragic story of the failure of the educational side of our social system.

RESPONSIBILITY OF MEDICAL PROFESSION FOR CONSERVATION
OF HEALTH

It is an important truth that nothing stands alone in this world, not even a medical association. We go up or down together. We are here today not solely because we are physicians, but also for the reason that society created us as a profession because it needed us, and we are therefore always serving its purpose. Our most private work is really a social and public work, so that in all we do we are going on the errands of society. Each one of us is an essential part of this moving human order that keeps society together and holds humanity to its sober tasks.

The world is passing into a new era. Though wars cease and the harsher strifes of men may disappear, the old struggle will go on under other forms which in the long run will be quite as destructive. Again the final test is the individual test, for in last analysis the reserve power of a nation rests on the ability of men and women to endure stress.

We cannot say that race decay has not already begun among the people of this country. There is much evidence that it has begun. No race nor nation, not even our own, can ever be free from the conditions that produce degeneracy. These are inherent in every form of human organization. They are all the more dangerous for being subtle and obscure in operation. They may act through generations lowering the resistances of individuals and families, and in widening circles a nation becomes involved.

Every medical association and every society should do systematic educational work with the public in the interest of a human conservation program. Medical men should not be willing to leave to others the credit or the task of leading in this movement, which is specifically a social duty of physicians. The profession owes it to society and to its own character as a scientific and progressive body to demand that the health and physical vigor of the men and women of the nation be cared for by the nation, and raised to the highest standard. For this, or any association of specialists, to take official notice of this subject, I realize would be a departure from custom. Departures from custom, new activities, are generally highly beneficial. They furnish stimulating experiences and a new form of spiritual exercise that is needed by the best of men and even by associations. They take the individual into bracing altitudes; they have been tonics to the social body in many an invalid period. All advance has been due to departures, that is, to new views, and new ideals that in all ages have set men's thoughts in higher and better ways. It has been such departures that have roused the inquiring, pioneering spirit that has led to great discoveries and kept men moving toward new horizons.

In this period of industrial and social reconstruction when all interests are becoming intertwined, when human interspaces grow smaller, and new relations create problems that change our views of life and society, it is necessary that the medical profession also make certain readjustments, that it annex human interests to its older activities, if its members are to maintain its fine traditions and keep step with social progress.

All professional life is beset by the danger of a certain bias which habits of work in any special line tend to create. We humans are made of malleable material and we are likely to be moulded to a rather definite shape by our occupation, all the more likely because the process is unconscious. It is not easy to resist the common tendency to become set in certain habits and ways of thinking, with the result of lessening of general interests, a limiting of intellectual curiosity and growth, which cause many men to atrophy on some of the best sides of character.

Society needs now, and needs more than ever before, men who answer to that severest test of character—the ability to grow. Not the linear growth that is in the direct way of men's daily work and which is a relatively easy process; men are needed who are capable of lateral growth, who recognize the new human borderland of interests that increasingly demand attention. It needs men who take the social view of their work, who understand the individual and social value of life and see the importance of increasing that value.

The people of this country do not appreciate the need of human conservation; they have not been told as only doctors could tell them that it is necessary and urgent. Shall we wait and allow society finally to lead, or shall we take the wider view of our mission, and become the pioneers of a great constructive movement?

And now permit me, gentlemen, to indulge my fancy.

Fifty years from this time a medical historian will refer to this Congress and the proceedings of this section. To make the record complete he may mention the poor performance of this presidential hour. I read what he will write: "At the opening of the neurological section the president omitted the subject of neurology and spoke on human conservation, though he did this rather apologetically, as though he were afraid of offending someone. Being, however, an elderly man and evidently a person of good intentions, he was listened to politely.

"After that time the medical profession went far beyond the feeble suggestions of the speaker, of 1919, and the American Neurological Association in particular recognizing the importance of medical sociology made itself famous by developing a plan that has achieved

astonishing results in fifty years. Briefly, the physique of the American people has been recreated since that time and they now are known as the most vigorous and virile and efficient nation in the world.

"The writer regrets to say that, after careful research, he has failed to find any record of the name or the fame, if he had any, of the president of the American Neurological Association of 1919."

CEREBROSPINAL FLUID IN EXPERIMENTAL COMPRESSION OF THE SPINAL CORD *

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In 1903, Froin¹ published cases of compression of the spinal cord due to a variety of causes but especially to meningitis; spinal fluid obtained by lumbar puncture was yellow, clotted rapidly, and contained an excess of protein. Since then this fluid has been called the *syndrome de coagulation massive et de xanthochromie*. It was subsequently found in a much greater variety of conditions, but all exhibited the common characteristic of cord compression.

In 1910, Nonne² called attention to the significance of a moderate to considerable increase in protein content of the spinal fluid, without xanthochromia, increase of cells or clotting. This type of fluid was frequently found in the presence of cord compression in a large number of cases published from Nonne's clinic by Raven.³

In 1916, the writer and Viets⁴ analyzed a number of personally observed cases, and from these and from the literature, concluded that the syndrome of Froin was almost pathognomonic of spinal cord compression, usually indicating either a considerable or a rapidly progressive compression. It was further concluded that Nonne's syndrome was suggestive of a lesser degree of pressure on the spinal cord, and so delicate a test was it found to be that in some cases in which cord compression was in doubt it was found to be the most significant sign obtainable.

EXPERIMENTS

Some experiments performed during the past year on cats are of interest on this subject.

1. *Method*.—Briefly, the method employed was to produce compression on the cord by means of paraffin in the epidural space; and

* From the Army Neurosurgical Laboratory, Johns Hopkins Medical School, Baltimore, Md.

* Authority to publish granted by the Board of Publication, Surgeon-General's Office.

1. Froin: *Gaz. d. Hôp.* **76**:1005, 1903.

2. Nonne: *Deutsch. Ztschr. f. Nerven.* **40**:161, 1910.

3. Raven: *Deutsch. Ztschr. Nerven.* **44**:380, 1912.

4. Ayer, J. B., and Viets, H. R.: Spinal Fluid Findings Characteristic of Cord Compression, *J. A. M. A.* **67**:1707 (Dec. 9) 1916.

subsequently to examine the spinal fluid above and below the area of compression.

After some practice it was found to be possible to introduce an 18 gage needle into the epidural space at lower thoracic levels without piercing the dura. Through this needle, paraffin with a melting point of 55 C., was injected; usually 1.5 c.c. being employed. A little carbon flour was added to blacken the paraffin to aid in its subsequent detection. At desired intervals thereafter spinal fluid was obtained by lumbar puncture and by occipito-atlantoid puncture of the cisterna magna;⁵ thus an opportunity was given to compare the fluids above and below the area of compression. Tests were performed as follows: Naked eye examination for color and clotting; precipitation with 95 per cent. alcohol for proteid, using similar quantities of spinal fluid from upper and lower subarachnoid space; cell counts and differential counts frequently. Unfortunately, the amount of fluid obtained is too little for determination of pressure or for quantitative protein tests.

The method is of advantage in giving compression of a more or less uniform degree, and in affording an opportunity to compare the fluid above and below this compressed area at known intervals of time. A disadvantage of the method is that it provides a type of cord compression not likely to be met with in man. A more important disadvantage is that after compression there is so little fluid in the lumbar sac, and that under such slight pressure, that lumbar puncture frequently yields only a drop or two.

2. *Symptoms.*—On coming out of ether, cats subjected to epidural injections usually show weakness in one or both hind legs, remaining in crouched attitudes and frequently dragging one leg. Seldom is complete paraplegia seen immediately after injection. On the day following, symptoms are usually more marked, with evident weakness of both hind legs, though rarely to the same degree. Seldom is the paralysis complete at any time in either limb, though infrequently complete paraplegia occurs. Sensation from pinching the leg or pricking the skin is almost always preserved.

Subsequently the paraplegia persists for weeks or tends to improve; at times, great improvement is seen. Owing, however, to the paralyzed condition it is difficult to keep these cats in good general condition and they frequently die in one or two weeks.

3. *Pathologic Findings.*—Following injections as outlined, the blackened paraffin is found in the epidural space closely molded to the

5. Wegeforth, Ayer, and Essick: Am. J. M. Sc. 157:1919.

dorsal aspect of the dura to a depth of from 1 to 3 mm. and extending along the dura for a distance of from 5 to 9 cm. Laterally, it thins out and is seldom seen over the ventral surface.

On removal of the paraffin, the cord, as seen through the dura, appears a little swollen, as indicated by its size and the loss of its finer markings. It is of a brownish tint and vessels appear, in the recently injected cases, abnormally prominent, not only at the site of compression but in the lumbar region below.

Microscopic examination shows at the levels of compression a mild cellular infiltration of the meninges. In recently injected animals this consists of polymorphonuclear cells, together with considerable serum and some free blood; in long-standing cases the exudate is made up of large mononuclear cells, some of which are phagocytic, together with lymphocytes and plasma cells. In neither type are organisms seen. The spinal cord shows no distortion, but may appear a little large. It seldom presents abnormality of gray or white matter as seen by hematoxylin and eosin or toluidin blue staining methods. Neither in gross or on microscopic examination is there evidence of burning of the tissues from hot paraffin.

4. *Examination of the Cerebrospinal Fluid.*—In nine cats in which paraffin was successfully injected, spinal fluid examinations showed in lumbar fluid the following:

GROUP 1.—Three cats show the Froin syndrome (in part or complete).

GROUP 2.—Four cats show the Nonne syndrome.

GROUP 3.—Two cats show normal fluids.

Spinal fluid above the area of compression, obtained by occipito-atlantoid puncture at the same time, invariably gave normal or relatively normal findings.

GROUP 1.—All of the cats showed almost complete paraplegia within twenty-four hours after injection; and punctures were made within forty-eight hours. The following protocol is from one of this group:

CASE 1 (1064).—Aug 14, 1918—11:20 a. m.: 1.5 c.c. paraffin (melting point 55 C.) injected into lower thoracic epidural space. Five p. m.: almost total paralysis of hind legs; cries out if tail is pinched.

August 15—9 a. m.: Can just waddle with hind legs. Reflexes lively.

Lumbar puncture yields two drops of yellow fluid which clots in the needle. With stylet a little is forced into 0.5 c.c. 95 per cent. alcohol, and a considerable white precipitate forms.

From *cisterna magna* is obtained 1 c.c. of slightly blood tinged fluid which does not clot and gives only a faint precipitate with alcohol. Both fluids show a few polymorphonuclear leukocytes and large mononuclear cells.

Sacrificed same day. Gross examination shows an almost continuous layer of blackened paraffin over dorsal aspect of dura from C₁ to L₂; paraffin appears

thinned out over lateral aspects of dura and only a little appears ventrally. The cord is obviously under tension as shown by transverse depressions corresponding to vertebral contours. The color is normal except for a few small hemorrhages, superficial but under dura. Microscopic examination shows a well marked but moderate polymorphonuclear exudate in the meninges, together with some serum and some free blood. No organisms. In the cervical region above the paraffin there is a very slight polymorphonuclear infiltration of the meninges. Cord substance appears normal.

The following case is of greater interest because the cat lived for nine weeks and numerous tests were made upon the spinal fluid.

CASE 2 (1065).—August 14—11:30 a. m.: Cat injected with 2 c.c. of paraffin in lower thoracic epidural space. Five p. m.: Partial paraplegia. Next day: Can just walk, hind legs very stiff. Tests of spinal fluid today show the Froin syndrome. (Compare table.) For nine weeks animal continued to show an awkward and stiff gait until, following ether and puncture, it died on October 17.

RESULTS OF TESTS OF THE SPINAL FLUID

Date	Lumbar Puncture				Occipito-Atlantoid Puncture			
	Amount of Fluid	Character of Fluid	Clot	Proteid	Amount of Fluid	Character of Fluid	Clot	Proteid
August 15	2 drops	Not determined	Clotting in needle	++++	1 c.c.	Blood tinged	No clot	+
September 7	0.5 c.c.	Slightly blood tinged	No clot	+	0.5 c.c.	Clear colorless	No clot	Slt. +
September 11	0.3 c.c.	Slightly blood tinged	No clot	++	Clear colorless	No clot	Slt. +
September 17	1 c.c.	Bloody fluid	No clot	++	Clear colorless	No clot	Slt. +
September 26	0.5 c.c.	Clear colorless	No clot	+	0.5 c.c.	Clear colorless	No clot	Slt. +
October 5	1 c.c.	Almost clear	No clot	+	1 c.c.	Almost clear	No clot	Slt. +
October 17	0.8 c.c.	Almost clear	No clot	Slt. +	0.5 c.c.	Clear colorless	No clot	Slt. +

Tests as follows: The amount of proteid is expressed relatively; "slt+" represents normal precipitation with alcohol—all others pathologic, ++++ being a massive precipitation.

At the time of obtaining the last fluids, with both needles in situ a little Locke's solution was introduced into the cistern, and with the slightest pressure it appeared promptly at the lumbar needle, thus showing at this time the continuity of the subarachnoid space.

Examination shows paraffin mass extending over the dorsal aspect of the dura from C₄ to filum. The dura is nowhere abnormally tense, but root markings as seen through it are a little obscure. There is no distortion of cord. Microscopic examination shows arachnoid membrane seemingly adherent to inner surface of dura. In the arachnoid meshwork are numerous mononuclear cells. In the gray matter of cord an occasional phagocytic cell is seen, but nerve cells and white matter appear otherwise normal.

These two cases show early the Froin syndrome in the lumbar fluid. In the latter case, subsequent examinations show a change to the picture described by Nonne, and later to normal. Cells were studied in the above fluids; during the earlier punctures a number of polymorphonuclears and lymphocytes was found, both above and below the area of compression, but later disappeared almost entirely from both fluids. Except where definite blood contamination was noted, red cells were not conspicuously present.

GROUP 2.—Of the four cats in this group, paralysis was less marked than in cases of Group 1, except in a single one. A protocol from this group is given:

CASE 3 (1098).—August 20—11:20 a. m.: Epidural injection of paraffin 0.3 c.c. followed in two minutes by 0.5 c.c. (Two separated injections given in an unsuccessful attempt to more closely localize the pressure.) Five p. m.: Somewhat spastic in hind legs—otherwise well. August 21: Does not use right hind leg much; it is kept forward under belly. Left hind leg stiff. 10:30 a. m.: Lumbar Puncture: Six drops of almost clear, colorless fluid shows numerous polymorphonuclear and mononuclear cells and a few red corpuscles. Proteid test strongly positive. Occipito-Atlantoid Puncture: 0.5 c.c. clear; rare cell; almost no proteid.

September 4—L. P. and O. A. P. fluids both clear. Protein—slightest possible trace in both. No white cells—rare red corpuscles in lumbar fluid. Animal remained spastic in hind legs until a third test was performed. September 10—L. P.: 0.5 c.c. slightly blood tinged fluid. Proteid—correct amount for blood present. O. A. P.: 0.5 c.c. clear; almost no proteid. September 11, sacrificed.

Gross examination shows paraffin in epidural space for a distance of 9 cm.; in its thickest portion 3 mm. deep. No microscopic examination.

This experiment shows in the condition of a moderate degree of paraplegia an initial lumbar fluid with marked but not excessive content, which subsequently becomes normal.

GROUP 3.—Two cats showed normal spinal fluids even with the pressure of paraffin in the epidural space. In both of them, however, paraplegia was of a slight degree; and in one the test was not made until long after the infection (on the 19th day), at a time when the fluid of similar cases was shown to have become normal.

SUMMARY AND DISCUSSION

It has been possible to inject with paraffin the epidural space in cats. Symptoms of partial transverse myelitis, manifested chiefly as incomplete paraplegia, have resulted.

In these cats the spinal fluid obtained from below the area of compression usually differs greatly from that above; the former shows almost constantly greater protein content, is usually scanty in amount,

at times is of a yellow color, and at times clots rapidly and completely; the fluid taken from above is uniformly and relatively normal.

These abnormal fluids are readily grouped into two classes, corresponding to the syndromes of Froin and of Nonne, recognized clinically to be characteristic of cord compression. As in man, the fluids which clot and contain the greater amounts of protein are found in the cases showing greatest symptoms of pressure. It has also been clinically observed that agents which rapidly produce compression of the spinal cord are those most likely to give the complete syndrome. In our experiments the injection of paraffin produces sudden compression; the well marked fluid changes recorded must have been promptly brought about, in that they were often demonstrated by tests made twenty-four hours later.

It may be thought that the abnormal findings are due to hemorrhage into the subarachnoid space incident to the injection. This is unlikely because normal fluids contaminated with an equal amount of blood either do not clot at all or yield at most a filmy clot; nor does the protein content in such fluids compare in amount with these. The protein associated with the mild aseptic meningitis present in these cases is also relatively insignificant, as demonstrated by the fact that the fluid obtained from the cisterna magna may contain white cells equal in number to those from the lumbar region, but shows only slight increase in protein. That these fluids may be in some manner caused by burning of the tissues from the hot paraffin is not the case, because a few experiments in which an amount of paraffin was injected insufficient to cause compression paralysis failed to cause changes in the lumbar fluid.

We see in some cases a well marked vascular engorgement of the pial vessels below the area of compression. We see also an abnormal amount of serum in the subarachnoid space at the level of compression. It is reasonable to think that transudation into the lumbar sac, as maintained by Mestrezat (page 448),⁶ is the pathologic process operative in the formation of these fluids so rich in protein.

CONCLUSIONS

1. Paraffin may be injected into the epidural space in cats, with resultant compression of the cord and symptoms of incomplete transverse myelitis.

6. Mestrezat: *Le Liquide Cephalo-Rachidien, normal et pathologique*, Paris, 1912.

2. Spinal fluid obtained from below the area of compression usually shows marked increase in protein content, at times is yellow and clots spontaneously. The greater the amount of protein the more likely is the fluid to clot.

Fluids obtained from the subarachnoid space above the area of compression are invariably normal or nearly normal.

3. The pathologic fluids obtained are entirely comparable with (1) the *syndrome de coagulation massive et de xanthochromie* of Froin, and (2) the syndrome of Nonne—both characteristic of spinal cord compression in man.

A CLINICAL SURVEY OF 415 INSTANCES OF BRAIN, SPINAL CORD AND PERIPHERAL NERVE INJURIES, AS SEEN IN OVERSEAS WOUNDED

WITH REPORTS OF SEVERAL UNUSUAL CASES *

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The cases constituting the basis of this report were studied at the U. S. Army Base Hospital, Camp Merritt, N. J., during the period from December, 1918, to March, 1919. As this institution evacuated the wounded to hospitals of the interior, the treatment given these patients during their brief stay was of a temporary nonsurgical nature. As a consequence, no therapeutic deductions were made from the treatment applied to this group of cases. The tables appended (Tables 1, 2 and 3) give a detailed analysis of the cases.

The 415 instances occurred in 328 patients, 76 of whom had two or more lesions. Of these multiple injuries, which occur with much greater frequency in lesions of the upper extremities, the commonest combinations are analyzed in Table 2.

These cases have been examined at this hospital, on an average of from three to five months after the original injury was sustained; therefore, a sufficient length of time has elapsed to allow of the completest evolution of the lesion. On the other hand, in certain brain and cord injuries (namely, aphasia and hematomyelia) a marked degree of recovery has been attained in that period of time; in such cases, history and field cards have corroborated the diagnosis.

PERIPHERAL NERVE INJURIES

In Tables 1 and 2 it will be noted that three columns have been drawn—one to indicate complete severance and loss of function of the nerve; a second, to indicate incomplete severance, contusion, concussion or commotio of the nerve, with incomplete loss of function, and a third, those cases of an incomplete nature, in which the pain or

* Authority to publish granted by the Board of Publication, Surgeon-General's Office.

TABLE 1.—STATISTICAL DATA ON 415 NERVE INJURIES

Nerve Involvement	Complete Involvement	Partial Involvement	Neuritis Causalgia	Total
Brachial plexus.....	2	7*	5	16
Root lesions.....	3	3
Cord lesions—				
Outer.....	1	1
Inner.....	5	2	..	5
Posterior.....	2
Median.....	35	27	8	70
Ulnar.....	43	24	8	75
Musculospiral.....	37	12	4	53
Radial (term. cutan. branch of musculo-spiral).....	8	8
Internal cutaneous.....	8	1	1	10
Circumflex.....	4	4
Musculocutaneous.....	3	3	..	6
Long thoracic.....	1	1
Nerve to rhomboids.....	1	1
Nerve to posterior axillary muscles.....	1	1
Total.....	152	78	25	256
Sciatic.....	12	4	5	21
Small sciatic.....	2	..	1	3
External popliteal.....	33	6	1	40
Internal popliteal.....	1	4	1	6
Posterior tibial.....	4	3	4	11
External cutaneous of thigh.....	3	3
Internal saphenous.....	2	1	2	5
External saphenous.....	1	1
Anterior crural.....	2	2
Total.....	58	18	16	92
Cranial Nerve Injuries—				
Olfactory.....	0
Optic.....	6	1	..	7
Oculomotor.....	2	3	..	5
Trochlear.....	0
Trigeminal.....	..	4	..	4
Nerve to external rectus.....	1	1	..	2
Facial.....	4	7	..	11
Auditory.....	2	2	..	4
Glossopharyngeal.....	0
Vagus.....	..	1	..	1
Spinal accessory.....	..	2	..	2
Hypoglossal.....	1	1	..	2
Total.....	16	22	..	38

* Commotio of plexus with residual lesions.

TABLE 2.—THE MOST COMMON COMBINATIONS IN MULTIPLE LESIONS

Nerve Involved	Complete Involvement	Partial Involvement	Neuritis Causalgia	Total
Median and ulnar.....	9	8	2	19
Median and musculospiral.....	1	1
Ulnar and musculospiral.....	1	1	..	2
Median, ulnar and musculospiral.....	5	3	1	9
Median, ulnar and internal cutaneous.....	2	2
Median and radial.....	3	3
Median, musculospiral and internal cutaneous.....	1	1
Median, ulnar and radial.....	1	1
Total.....	24	12	3	39

causalgia have been of predominating severity. In the column of "partial involvement" are also included those cases in which only the sensory or motor components of a mixed nerve have been involved.

In regard to peripheral nerve injuries, comparison of our statistics with those of recent English writers (namely, Burrow and Carter,¹ and Stewart and Evans²), reveals on the whole a close correspondence; certain discrepancies, however, require comment. These authors have found brachial plexus injuries more commonly than we have; namely, Burrow and Carter, 77 out of 1,000 cases, and Stewart and Evans, 61 out of 316 cases.

TABLE 3.—BRAIN INJURIES—SECONDARY TO FRACTURED SKULL

Brain Injuries—Secondary to Fractured Skull—	
Aphasias (motor and sensory) partial.....	7
Hemiplegias	4
Paraplegias	1
Astereognosis (injury to superior parietal lobe left).....	1
Occipital lobe injuries (with hemianopsia).....	2
Posttraumatic (fracture) psychosis.....	1
Total.....	16
Spinal Cord Injuries—	
Hematomyelia (concussion)	4
Cauda equina	3
Conus terminalis	1
Cervical sympathetic palsy.....	1
Segmental lesions:	
Cervical.....	1
Dorsal.....	0
Lumbar.....	1
Sacral.....	2
Total.....	13

In studying the histories of the severer injuries of the upper arm and shoulder, commotio of the brachial plexus has been noted very often. In fact, the great majority of men sustaining severe wounds of the upper arm and shoulder give a history of a fairly complete sensory and motor paralysis of the corresponding arm, occurring directly after the injury. This paralysis quickly improves; many finally achieve complete restoration of function, others show residual sensory and motor lesions.

In their combined total of 1,316 cases, circumflex nerve involvement occurred twice, whereas, in our series four instances are recorded, three of which occurred singly. Our one case of long thoracic nerve involvement (associated with a commotio of the brachial plexus) and our one instance of involvement of the nerves to the rhomboidei, find no parallel in their series. Of our ten lesions of the internal cutaneous

1. Burrow, J. LeF., and Carter, H. S.: Preliminary Note on Investigations on 1,000 Consecutive Cases of Peripheral Nerve Injury, *Brit. M. J.* 2:535 (Nov. 16) 1918.

2. Stewart, Purves, and Evans, Arthur: *Nerve Injuries and Their Treatment*, Oxford Medical Publications, 1916. (These authors give statistics on 316 cases of nerve injury.)

nerve only one occurred singly, that is, unassociated with the other lesions. Of the six musculo-cutaneous lesions, five occurred associated with other nerve injuries.

LESIONS OF THE LOWER EXTREMITIES

In contrasting lesions of the lower extremities, interesting comparisons arise in regard to sciatic nerve injuries. Burrow and Carter record 121 out of 1,000 cases; Stewart and Evans, 21 of their 316 cases; we have had 21 in our series of 415 cases. In regard to external popliteal involvements, the former English authorities record 97 out of their 1,000 cases, the latter 26 of their 316 cases; we have had 40 instances. Many sciatic injuries quickly resolve themselves into partial or complete involvements of the external popliteal branch, a fact commented on by Stewart and Evans. The internal popliteal escapes with an incomplete involvement, which, as emphasized by Hammond,³ shows a marked tendency to recovery without special treatment.

As a consequence, we believe that not a few of our external popliteal involvements were really incomplete sciatic lesions in the beginning.

The English authors quoted do not record any instances of involvements of the external cutaneous nerve of the thigh; we have observed three such lesions. On the other hand, to the ten instances of anterior crural nerve involvement which their series of 1,316 cases reveal, we can only add two incomplete involvements with neuritis.

While neuromas on the whole are not frequently met with in peripheral nerve injuries, the great majority that are felt occur in the course of the median and ulnar nerves in the upper arm; a few are palpated along the course of the sciatic nerve in the thigh.

The cranial nerve injuries tabulated require no particular comment.

The clinical and symptomatological minutiae of the various nerve injuries are omitted in this paper; for such detailed clinical descriptions reference may be made to the excellent works of Mme. Athanassio Benisty,⁴ Tinel,⁵ and Stewart and Evans.²

BRAIN INJURIES

It is beside the purpose of this paper to go into the subject of brain injuries, concerning which such a vast literature has already arisen.

3. Hammond, T. E.: The Involvement of the External and Internal Popliteal Nerves in Lesions of the Sciatic Nerve, *Brit. M. J.* 1:397 (April 6) 1918.

4. Benisty, Mme. Athanassio: *The Clinical Forms of Nerve Lesions* (English translation), Military Medical Manuals, University of London Press, Ltd., 1918.

5. Tinel: *Les Blessures des Nerfs*, Masson et Cie, Paris, 1916.

Two very interesting cases coming under our notice, however, deserve mention.

ILLUSTRATIVE CASES

CASE 1 (J. H., Infantry Sergeant).—*Diagnosis: 'Cerebral concussion (laceration?) following severe head trauma (fracture of the base of the skull?)*.

The prolonged period of amnesia—the auditory aphasia, the sudden and peculiar changes in conduct characterized by the attacks of antagonistic behavior and negativism (posttraumatic psychosis), together with the partial loss of memory, the attacks of vertigo, tinnitus, restlessness, severe headaches, and the concentric contraction of the visual fields, all denote a diffuse cerebral injury involving especially the left temporal and occipital lobes, and optic paths. Such a complex grouping of symptoms and signs renders the case of especial interest.

History.—The patient has served ten years in the U. S. Army with a good record. He was admitted to the U. S. Army Base Hospital, Camp Merritt, N. J., Feb. 11, 1919; when examined, February 19, he talked freely and gave a fairly connected history of one year's foreign service. In July, 1918, he was gassed twice. On July 15, 1918, at Chateau-Thierry during a bombardment he was severely shocked with high explosives, and was "blown up twice"; during the second explosion he came down and hit his head, but was not rendered unconscious; he was dazed and suffered from loss of speech and hearing at this time. Later that day while being taken prisoner, he was severely beaten about the head with the butts of the German guns, and from the history of others, bled from nose, mouth and ears, at this time. The patient has no recollection of anything that occurred from July 15 to early September, but the history shows that he had numerous outbreaks of irritability while in a German prison camp, getting into fights with the German prison officers and attendants.

The first indication of return of memory occurred early in September, 1918; he remembers being able to talk, "but not well"; he could see, but the letters had to be very large. At that time he was unable to understand spoken language; it was necessary to put everything in writing. Since that time the patient has gradually improved, but he has had attacks of loss of memory, preceded by vertigo, constant ringing, loud noises in both ears, mental confusion, restlessness, severe headaches, and throbbing at the temples, terminating in profuse sweating and marked weakness. Some of these attacks were associated with mental derangement. On Feb. 19, 1919, the patient while being subjected to a Bárány test, had a violent propulsive seizure, and would have fallen heavily had he not been caught. There was no past-pointing. This reaction seemed to upset him greatly, and he felt very badly for several days.

Neurologic Examination.—Motor: Negative. Gait negative.

Sensory: Negative.

Reflexes: Knee jerks, double plus; Achilles, plus; arm reflexes, plus.

Cranial Nerves: Pupils equal, react to light and accommodation. Slight nystagmus to left. Fundi: pale, outline distinct, no signs of neuritis. Hearing, apparently diminished, but the behavior of the patient suggested at this time that his difficulty in understanding was an aphasic difficulty rather than due to an inherent defect in hearing. After answering numerous questions, the patient's behavior underwent a remarkable change; he became suddenly indifferent, refused to answer, looked down, and became sullen and irritable. Further examination discontinued until February 24, when the patient was seen in the ward smoking a cigar. He refused to talk, dropped his head, seemed

quite annoyed, and was markedly negativistic. After several attempts to get him to talk he arose from his chair, advanced toward the examiner, and made an attempt at assault. He was finally quieted and told to sit down, which he did. He rested his head on a table, and then began to move it with a jerky, lateral movement, becoming quite prostrated.

On March 1, the patient was much improved, talked freely, stating that he had no knowledge of the previous visit of the examiner, in fact, he said that he had never seen the examiner before. He was surprised when told that he exhibited an antagonistic attitude on the last examination. He stated that he was subject to such outbreaks, and that "his nerves were all gone," that he cannot remember, and does not sleep well.

Examination March 6: Patient not so well, spoke freely; he remembered the examiner, and some of the conversation of the last visit. Orientation, judgment and insight good. Marked concentric contraction of visual fields; photophobia; marked tinnitus complained of, worse in left ear. Hearing not diminished. His vocabulary is very limited, his speech slow, and he mixes his words. Before answering, he repeats the questions asked of him. No dysarthria. Memory: very distinctly amnesic, with complete blotting out of certain memories. Patient shows evident psychic exhaustibility; at first he conversed very well, but toward the end of the examination, he exhibited marked evidences of mental fatigue. The patient himself notices this, remarking, "if you keep at me too long, I can't think at all."

Roentgenogram.—This reveals no evidences of fracture of the skull.

Results.—The general behavior of the patient indicates that he has considerable difficulty in understanding spoken language. He makes no attempt to reply to questions until he advances close to the examiner, and watches the latter's lips very carefully; he then replies only after repeating the question asked. (This represents an effort to correct his auditory aphasia.) This is in contrast to his spontaneous speech which is comparatively fluent and connected. Patient's emotional behavior is now of a distinctly humorous turn.

CASE 2 (W. G.).—Diagnosis: Injury to cervical cord following fracture of left transverse processes of sixth and seventh cervical vertebrae; cerebral thrombosis, left, following ligation of external carotid artery in the neck (thrombus, ascending in the internal carotid artery).

This case is of especial interest in the unusual combination of lesions presented. Due to the cervical cord injury, we have a flaccid spinal monoplegia (left upper extremity) with sensory changes and a left cervical sympathetic paralysis with ptosis and miosis. On the other hand, incident to the cerebral thrombosis, and involvement of the vascular channels supplying the left sensory-motor areas, and motor speech center of Broca, there developed a motor aphasia, with right sided hemiplegia, hemihypesthesia, hemihypalgesia, right sided loss of muscle sense and partial astereognosis.

History.—A cerebral hemiplegia (with motor aphasia) on one side, and a spinal monoplegia with associated sympathetic palsy on the other, is certainly a rare combination. Shrapnel injury of left side of neck was sustained Sept. 28, 1918. Such a severe hemorrhage occurred at the site of injury that ligation of the external carotid artery on the left side became necessary. Two hours after the operation a right-sided hemiplegia and hemianesthesia supervened, more marked, however, in the upper than in the lower extremity. Loss of speech, with loss of power and numbness of left arm and ptosis of left upper eyelid, and miosis of left pupil also appeared.

Neurologic Examination (Dec. 15, 1918).—This revealed the following:

Motor: There is weakness of right arm, and to a lesser extent, the right leg. A marked weakness exists in the left arm, but none in the left leg. Grips: left, weak; right, weak (but stronger than left). No atrophy of definite muscle groups. Gait: slightly hemiplegic on right. Some spasticity of right hand and arm. Limitation of forward movements of cervical spine.

Sensory: Hypesthesia, hypalgesia, loss of muscle sense, and partial astereognosis noted in right upper extremity, more marked in the distal part. Sensation of right lower extremity, normal. Hypesthesia and hypalgesia present in the upper extremity—more marked than on right side. There is tenderness of seventh cervical spine.

Reflexes: Knee jerks: right, double plus; left, normal. Achilles: right, active; left, normal. Babinski present on right. Arm reflexes: right increased; left, normal.

Cranial Nerves: There is a left partial ptosis, associated with a pupillary miosis. Fundi: white patches of choroidal atrophy present in both eyes adjacent to the disks. Speech: A distinct paraphasia is present which apparently is the residuum of the previous motor aphasia. There is considerable difficulty in pronouncing "g's," "p's," and "c's" (dysarthria). Memory shows marked defect.

Roentgen-Ray Findings (Dec. 28, 1918).—Loss of bony tissue in the region of the left transverse processes of the sixth and seventh cervical vertebrae.

SPINAL CORD INJURIES

As will be noted in Table 3, four cases of hematomyelia are recorded. In view of the late work of H. Claude and J. Lhermitte,⁶ the propriety of designating these cases as "hematomyelia" is seriously questioned. These authors, in a recent article describing similar injuries in the French soldier, emphasize the factor of concussion in the production of the pathology and classify their cases as (a) *Indirect Conclusion*: This results from the explosion of shells of large caliber at a distance producing marked variations in atmospheric pressure; the individual affected is not struck by the missile. (b) *Direct Concussion*: (1) Immediate—in which the missile passes through the body of the vertebrae, or fractures the transverse or spinous processes, and (2) mediate—in which the missile passes through perispinal tissues or impinges on neighboring bones, so producing concussion of the cord tissues.

Both in the mediate and immediate types of direct concussion, the clinical syndromes produced are identical; all degrees and grades of pathologic phenomena may be brought about in these two types of direct concussion. The French authors⁷ emphasize the absence of

6. Claude, H., and Lhermitte, J.: Les Commotions directes de la Moelle Epiniere, Presse Med. 26:514 (Oct. 7) 1918.

7. Claude, H., and Lhermitte, J.: Etude clinique et anatomique de la Commotion directe de la Moelle Epiniere, Ann. de Med. 2:479 (Nov.) 1915.

hemorrhage in these direct concussion cases, and refer to the pathology in the cord as one of necrosis of nerve cells, axis-cylinders, and myelin sheaths, affecting the white fibers much more than the gray matter, and involving the central canal and posterior roots often to a remarkable degree. Neuroglia, vascular and other connective tissue elements show active proliferation. The following patients (Cases 3 to 8) are examples illustrative of the direct type of concussion, although tabulated by us in part as instances of hematomyelia. It will be noted that Cases 3, 4, 5 and 7, represent the immediate types of direct concussion, whereas Cases 6 and 8 illustrate the mediate variety.

CASE 3 (I. S.).—*Diagnosis: Injury to fifth dorsal segment of spinal cord following fracture of lamina of fifth dorsal vertebra and the right transverse processes of sixth and seventh dorsal vertebrae.*

The trauma produced a paraplegia with bladder and rectal disturbances, which recovered to a remarkable degree and sensory changes, which were of the dissociated type seen in syringomyelia. This case not only illustrates the great degree of recovery attainable in these cases, but also shows how the brunt of the pathologic process may be borne by the tissues about the central canal of the cord.

History.—Gunshot wound, July 21, 1918, penetrating right side and back of chest with comminuted fracture of sixth and seventh ribs, with injury to fifth and sixth dorsal vertebrae.

Previous examinations made abroad give the following data (from soldier's field card) July 29, 1918: "Paraplegia: right side, complete; left, nearly so. Roentgen ray reveals foreign body above left scapula, nearly subcutaneous. Fracture of eighth rib at entrance wound; fifth, sixth and seventh ribs near the spine. Fracture of transverse process, right side dorsal sixth and seventh vertebrae. Fracture lamina of fifth dorsal vertebra. Bladder and rectal control not lost, but disturbed. Hydropneumothorax present."

Aug. 18, 1918: "Power returning in legs; bladder and rectum the same. Incontinence of urine and feces, anesthesia of urethra, bladder, and rectum of two months' duration."

Neurologic Examination (Feb. 20, 1919).—This revealed the following:

Motor: No weakness of lower extremities. Gait, normal.

Sensory: Complete thermo-anesthesia, hypesthesia and analgesia below the level of fifth dorsal cutaneous segment with retention of deep sensation, except over buttocks, where there are two areas (islands) of retained heat sensibility. (Dissociation of sensation.)

Reflexes: Both knee jerks quite exaggerated. Patellar clonus on left side. No ankle clonus. Babinski and Oppenheim present on both sides. Although analgesic, when stuck with a pin, a marked motor reflex occurs in extensor muscles of both thighs, showing irritability of pyramidal tracts. A very slight Romberg. Marked *cutis anserinus* present, ephemeral in character.

Cranial Nerves: Negative.

Roentgen-Ray Findings (Feb. 23, 1919).—There is a comminuted fracture through the sixth and seventh ribs near the vertebral ends on the right side, with fractures of the transverse processes of the fifth and sixth dorsal vertebrae. There are some small fragments of metal near the fifth rib.

CASE 4 (E. M.).—*Diagnosis:* Injury of the cauda equina, following fracture of the fifth lumbar vertebra.

This produced a paraplegia with bladder and rectal disturbances, pains and tenderness along the sciatic nerves (descending neuritis) and a bilateral segmental loss of sensation in the entire sacral distribution. In this case the recovery attained is not as pronounced as in the preceding one. The presence of tenderness along the left sciatic nerve indicative of a descending neuritis is of especial interest.

History.—Gunshot wound in lower back sustained Oct. 14, 1918, followed by paralysis and numbness of lower extremities, retention of urine and marked constipation, associated with anesthesia of rectum, urethra, and bladder. After a period of two weeks, partial return of vesical and rectal power together with return of power in flexors and extensors of both thighs occurred; return of sensation also made its appearance on anterior aspect of both thighs. With numbness and paresthesia, shooting pains radiated down both sciatic nerves, the latter persisting on the left side. Movements of toes and dorsiflexion of feet, especially right, then partially returned. Loss of power of erection persisted for three months.

Neurologic Examination (Feb. 14, 1919).—This revealed the following:

Motor: Atrophy of glutei, calf, anterior tibial and peroneal muscles on both sides; weakness more marked on left side. Bilateral weakness of dorsi-flexors of feet especially marked on left side, producing partial bilateral "drop-foot." Gait, typically "steppage."

Sensory: Anesthesia and analgesia in cutaneous distribution of all sacral segments; anesthesia of urethra, bladder, and rectum. The first and second sacral segmental areas show irregular patches of return of sensation, more marked on right side; left sciatic nerve tender.

Reflexes: Abdominal, active and equal. Cremasteric, active and equal. Knee jerks, right, active; left, diminished. Achilles, both lost. Upper extremities normal.

Roentgen-Ray Findings.—The fifth lumbar vertebra shows a fracture of the left side of its body with a loss of bony substance $2\frac{1}{2}$ by $\frac{1}{2}$ cm., without dislocation or compression.

CASE 5 (A. L.).—*Diagnosis:* Injury to cauda equina following fracture of the fifth lumbar vertebra producing a syndrome of greater severity than in the instance of the preceding case. Paraplegia, muscular fibrillations, bilateral segmental loss of sensation in the sacral distribution of an asymmetrical nature, associated with trophic disturbances in foreskin and glans penis, are present. The descending neuritis in the sciatic nerves is very severe, occupying a prominent place in the clinical picture.

History.—Sustained a shrapnel wound of the fifth lumbar vertebra in September, 1918, following which he developed paralysis and numbness in the entire left leg and thigh, muscular twitchings in both legs and thighs, and retention of urine and feces. About ten days later, urinary incontinence took the place of retention. He gradually improved, was able to walk, and was sent back to this country.

Neurologic Examination (Feb. 1, 1919).—This revealed the following:

Motor: Atrophy of the muscles of both buttocks (glutei) especially left; atrophy of left hamstring muscles. Marked muscular twitchings in muscles of both thighs and legs, and also to a lesser extent in the muscles of the arms and forearms.

Sensory: Touch, deep pressure, pain and temperature absent in the cutaneous distribution of the left sacral 2-3-4-5 and coccygeal 1 segments, and in the cutaneous distribution of the right sacral 3-4-5 and coccygeal 1 segments. This involves the penis and the left half of scrotum, which are anesthetic and analgesic. The right half of the scrotum is hypesthetic. Trophic disturbances are manifested in foreskin and glans penis. There is anesthesia of rectum, bladder and urethra.

Hyperesthesia and hyperalgesia of skin along spinal column up to the level of the cervical spine. Sharp pain radiating down both thighs and legs into soles of both feet occurs, much more severe on the left, on which side it is associated with marked dysesthesia. Pressure along left calf and thigh is productive of severe pain.

Reflexes: Knee jerks, equal and active. Both Achilles absent. Upper extremities exaggerated.

Cranial Nerves: Negative. Incontinence of urine and persistent constipation present.

Roentgen-Ray Report (Feb. 4, 1919).—There is a piece of shrapnel $2\frac{1}{2}$ cm. long and 1 to $1\frac{1}{2}$ cm. wide resting above the left sacro-iliac joint. The bone is apparently normal beneath. Another small fragment 1 cm. long and $\frac{1}{2}$ cm. wide is located 1 cm. from the left border of the fifth lumbar and first sacral vertebrae. There has been some bone destruction on the left border of the body of the fifth lumbar vertebra, and a piece of bone about 1 cm. long is missing. This destruction of bone lies almost directly above the location of the smaller foreign body; there has been a deposit of inflammatory tissue about this area. There is no other evidence of fracture of this vertebra and no compression present.

CASE 6 (R. P.).—Diagnosis: *Injury to conus terminalis producing the "conus terminalis syndrome."*

The bilaterally symmetrical ano-genital anesthesia (sacral fourth and fifth segments) with complete retention of urine, occasional fecal incontinence, sexual impotence, together with the absence of pain and quick disappearance of fibrillations make for a clean cut clinical picture.

History.—Sustained gunshot wound Sept. 6, 1918, entering left hip, passing across region of first and second sacral vertebrae, emerging at right hip. At the time of injury, partial paralysis of both legs, with muscular twitchings of lower extremities—retention of urine, loss of rectal control, and loss of power of erection occurred.

Neurologic Examination (Dec. 31, 1918).—This revealed the following:

Motor: Walking slow and difficult; gait, straddle-legged and paretic, though no actual paralysis. Muscular twitchings have disappeared.

Sensory: Anesthesia and analgesia of penis, scrotum, perineum, bladder, urethra and rectum; also, a band of anesthesia extending around the lower abdomen and the inner aspect of thighs, above which was an area of hyperesthesia. The sensory changes are symmetrical. Except for constant backache, no pains are complained of.

Reflexes: Knee jerks, equal and active. Cremasteric, present, left greater than right. Abdominals, equal and active. Upper extremities, normal.

Cranial Nerves: Negative. Complete retention of urine, necessitating catheterization, present together with marked constipation, and occasional fecal incontinence.

Reexamination (Jan. 11, 1919): Examination at this time revealed:

Motor: No change.

Sensory: Area of anesthesia diminishing; and limited to the cutaneous distribution of the fourth and fifth sacral segments on both sides. Hyperesthetic areas above first lumbar regions still present.

Reflexes: No change.

Retention of urine, persistent constipation with occasional fecal incontinence, impotence, and anogenital anesthesia still present.

Roentgen-Ray Examination.—Reveals no injury of bones of lower spine.

CASE 7 (R. M.).—*Diagnosis:* *An unusual case of concussion (hematomyelia?) of cervical cord, produced by injury to the spinous processes of fifth and sixth cervical vertebrae.*

The involuntary, painful, muscular contractures of the fingers, the atrophy of the muscles of the upper extremities, the dysesthesiae and parasthesiae, and other sensory and trophic disturbances, all of a symmetrical nature constitute a very interesting and rare syndrome. These irritative manifestations are dependent, we believe, on lesions of the posterior roots and columns, pyramidal tracts and anterior columns of a partially destructive nature.

History.—Gunshot wound of neck sustained in October, 1918. The missile entered the left side of neck, passed horizontally across the median line, emerging at a corresponding point on right side, the scars of injury being on the level of the spinous process of the fourth cervical vertebra.

Following the injury, the patient suffered a complete motor paralysis of upper and lower extremities and a sensory paralysis of the upper extremities; the lower extremities gradually regained their motor power.

Neurologic Examination (Dec. 21, 1918).—This revealed the following: Marked tenderness to pressure over the fifth and sixth cervical vertebrae over which spinous processes there is a loss of the usual bony contour. The superficial fibers of both trapezii have been severed. Lateral movements of head restricted and painful. A marked, painful flexor contracture of fingers of both hands present, producing an involuntary clenching; the palms show as a consequence the imprint of the finger-nails. He complains of marked tenderness of skin of hands, of paresthesiae, and of severe painful cramps in muscles of hands.

Motor: Weakness of both upper extremities, more marked distally; especially flexors of forearm and intrinsic muscles of hands with consequent limitation of the finer movements of fingers and hands. Atrophy of muscles of both shoulders, arms, forearms and hands; grips, weak—left weaker than right. No fibrillary twitchings; a fine rapid tremor of hands is present; the contractures are noted above.

Sensory: Skin of hands very tender and intolerant to cold; areas of dysesthesia, hypesthesia, hypalgesia and thermohyperesthesia of hands and forearms. Trophic: skin pink, glossy and dry. Astereognosis: left complete, right partial.

Reflexes: Deep reflexes of lower extremities, normal. Upper extremities, left arm, increased; right arm, normal.

Cranial Nerves: Pupils contracted, outline regular, light reaction diminished. Fundi show marked congenital conus.

Roentgen-Ray Findings.—There is destruction of spinous processes of fifth and sixth cervical vertebrae. The bodies of the vertebrae are apparently normal. The ligamentum nuchae has been partially severed.

CASE 8 (A. W.).—*Diagnosis:* Injury to cervical and dorsal cord (cervical fifth to dorsal second segments) following gunshot wound of neck.

In this case there is no evidence of direct (immediate) injury to the vertebral column and yet the concussion of perispinal tissues was transmitted to the cord in such a violent manner as to produce a distinct segmental injury (C_5 to D_2) which manifests itself in the muscular atrophy of the left shoulder group, in a left sympathetic ptosis and myosis (which disappeared) and in a Brown-Séquard syndrome involving the left half of the cord in the cervico-dorsal region.

History.—Shrapnel wound of left side of neck sustained Oct. 1, 1918, situated behind the posterior border of sterno-mastoid muscle of left side, $3\frac{1}{2}$ inches below the tip of the mastoid process, producing loss of power of left arm, leg, and partial loss of power of right leg with loss of sensation of right side of body, including arm, leg, and trunk. He was not unconscious, but there was considerable bleeding from the wound. At this time there was also noticed a drooping of his left upper eyelid, and a contraction of the left pupil. Twenty hours after the injury the patient was operated on and a piece of shrapnel, with particles of clothing were removed; following the operation loss of voice and dysphagia appeared (injury of left recurrent laryngeal nerve) Five days later wound culture showed *Bacillus welchii* and nonhemolytic streptococci.

Patient progressively improved and was finally sent back to this country. He was admitted to U. S. Army Base Hospital, Camp Merritt, N. J., Feb. 21, 1919, suffering from marked dyspnea; examination then showed such edema of larynx and glottis that a low tracheotomy had to be performed; prompt relief followed.

Neurologic Examination (Feb. 23, 1919).—This revealed: A purulent discharge from the operative wound which now evidently communicated with the tract of the missile; subcutaneous emphysema noted in both supraclavicular spaces. Lateral movements of head limited and painful; tenderness over spinous processes of cervical vertebrae.

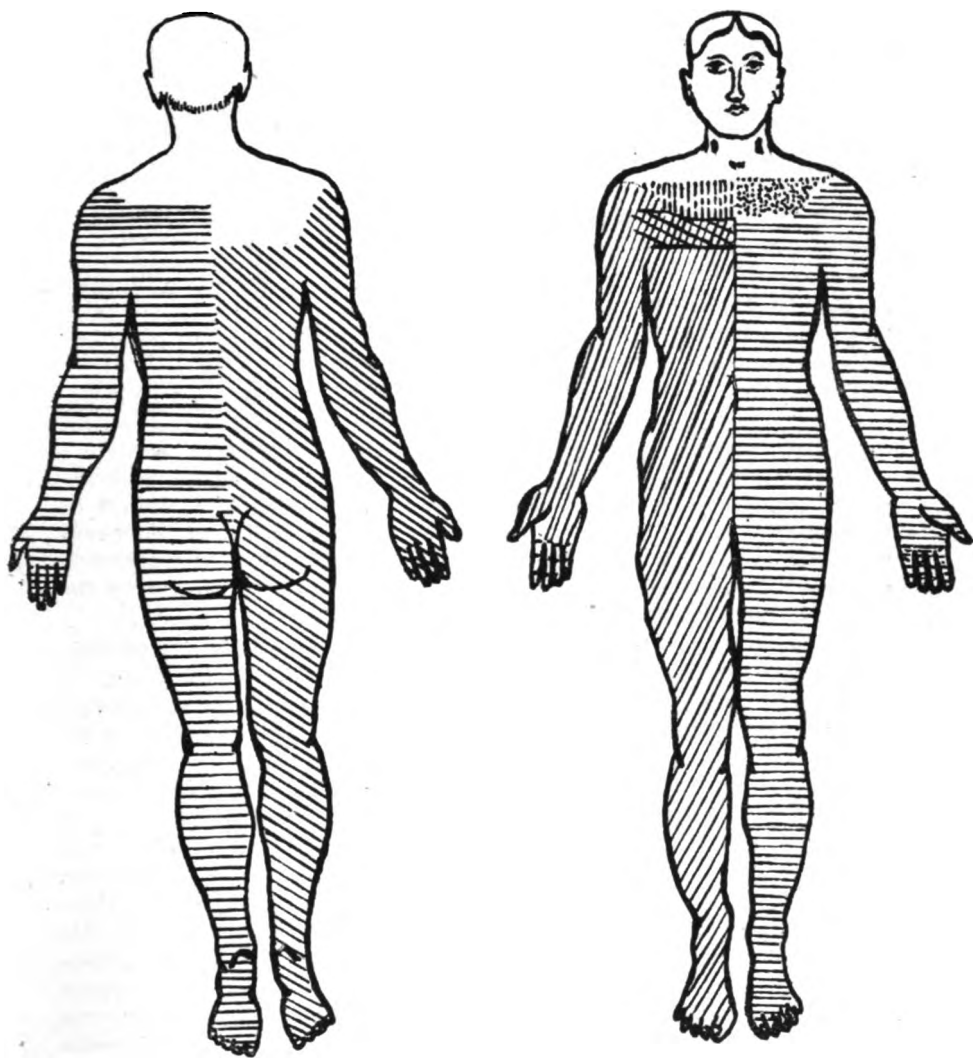
Motor: Weakness of left arm (especially muscles of shoulder group) and of left leg. Right upper extremity, normal; right lower, weak. Grips: left, weak; right, normal. Considerable atrophy of left supraspinatus, infraspinatus, deltoid, biceps, pectorales, trapezius, and to a lesser degree, of the muscles of left forearm and hand. Some weakness of both lower extremities, not enough to prevent efficient locomotion.

Sensory: Refer to the accompanying chart.






Reflexes: Knee jerks: right, active; left, double plus. Achilles reflex, right, active; left, double plus. Ankle and patellar clonus present on left, on which side there is also a Babinski. Biceps and triceps are normal on the right, but increased on the left. Abdominals and epigastrics, normal on the right, absent on the left.

Cranial Nerves: Negative. Left sympathetic ptosis and pupillary miosis now gone.

Roentgen-Ray Findings.—This was negative for bony changes in cervical spine (March 20, 1919). Pain and temperature sense have returned on the right side, down to the level of the fourth intercostal space.



Illustrating sensory changes in Case 4.

1.  1. Area of hypesthesia; deep sensation retained.
2.  2. Area of analgesia, thermo-anesthesia and hypesthesia; the hypesthesia is less marked on this side.
3.  3. Area of thermohypesthesia, hypalgesia and hypesthesia.
4.  4. Area of thermohypesthesia, hyperalgesia and hyperesthesia.
5.  5. Area of thermohypesthesia. Areas 3, 4 and 5 not well differentiated on back.

FUNCTIONAL CASES

Herewith are presented three cases of a functional nature, each peculiarly interesting in certain of its features.

CASE 9 (N. B. M.).—*Diagnosis: Concussion of lumbar cord producing a paraplegia with sensory changes and bladder disturbances.*

As the organic lesion was recovering, a functional condition developed, characterized by clonic tic-like movements of face and upper part of body, emotional instability, headaches, left sided hemianesthesia, hemianalgesia, and "glove and stocking" anesthesia and analgesia on the right side.

History.—This patient was "blown down" while in a barrage and fell headlong into a shell hole, producing marked hyperextension of his spinal column. Following this he was dazed and his lower extremities felt numb and paralyzed; he was unable to move them for about ten days, when motion gradually returned. Incontinence of urine was present and has persisted. It consists of an inability to control the flow. There is no retention or loss of bowel control. The patient also complained of a dull constant pain in the lower back, with radiations of a sharp nature encircling the abdomen, at the level of the iliac crests, and also stiffness of lower extremities.

Neurologic Examination (Dec. 19, 1918).—This revealed the following: Motor: Great difficulty in walking noted, due to pain and stiffness of back.

Sensory: Irregular areas of hypesthesia and hypalgesia over entire body; a band of hyperesthesia in lower dorso-lumbar region extends around the trunk.

Reflexes: Knee jerks, equal but diminished. Achilles, present and equal. Pupils, irregular—react to light and accommodation.

The spinal column is so rigid that he cannot bend over. He complains of marked restlessness, headache, vertigo, and poor vision.

Subsequent Examination.—More recently, since the latter part of December, 1918, clonic irregular, tic-like movements of neck and shoulders occur. These movements are much worse on emotional excitement, hearing noises, etc. He is still subject to headache (whereas formerly he was quite immune); he is emotionally unstable, weeping readily. His speech is quite hesitating, whereas formerly he was able to discourse freely. Facial grimaces and movements occur now, whereas formerly they did not. He complains of "specks" before his eyes, and cold feet quite frequently.

Ten years ago he had an attack of "nervous prostration," which lasted over a period of six months; he was confined to his bed for the first six weeks. The attack was ushered in by unconsciousness, associated with muscular movements of an involuntary nature on the right side of body, and partial loss of memory. He finally made a good recovery.

Reexamination (Jan. 22, 1919).—Examination at this point revealed the following:

Motor: He walks with a shuffling, spastic gait, holding his back quite rigid. No muscular atrophy or hypertonicity.

Sensory: Complete *left-sided* hemianesthesia and hemianalgesia.*

On *right side* anesthesia and analgesia of lower extremity up to groin, and of lower third of right forearm and hand. Tenderness in lower back.

* Along inner sides of both legs there remains a longitudinal, narrow area of retained sensation.

Reflexes: Knee jerks, both diminished. Achilles present and equal. No Babinski or Oppenheim.

Final Results.—Later reports from U. S. Army General Hospital No. 1, New York, to which this patient was transferred, indicate that he has almost entirely recovered from his functional disturbances.

CASE 10 (J. W.).—*Diagnosis: Hysterical monoplegia, with "glove" anesthesia and analgesia associated with a homolateral hemihypesthesia and hemihypalgesia following a gunshot wound of left forearm.*

This case illustrates the "fixing" of a hysterical monoplegia by a gunshot wound of the same limb.

History.—Gunshot wound of left forearm, $3\frac{1}{2}$ inches below the elbow, sustained July 21, 1918.

Neurologic Examination (Jan. 20, 1919).—This revealed a functional paralysis of all the muscles of the left forearm and hand; all electrical reactions preserved; no demonstrable organic nerve lesion. Slight degree of atrophy (disuse) in muscles of left forearm and hand. Complete "glove" anesthesia and analgesia to $1\frac{1}{2}$ inches above site of wound, associated with which there exists a left hemihypesthesia and hemihypalgesia.

CASE 11 (J. G.).—*Diagnosis: Hysterical "glove" anesthesia and analgesia following a trivial injury (gunshot wound) of the little finger of the left hand.*

The interest in this case centers about the fact that as the sensory changes began to disappear, the area of anesthesia remaining very closely simulated the cutaneous sensory distribution of the ulnar nerve in the hand.

History.—This soldier gives a history of having been gassed and "shell shocked" before sustaining a gunshot wound of the little finger of the left hand on July 15, 1918; this injury was very evidently of a trivial nature. The history shows that for six weeks after the injury there was a complete "glove" anesthesia and analgesia to above the left wrist. This gradually cleared up.

Neurologic Examination.—When examined by us Feb. 6, 1919, the functional area of anesthesia remaining very closely simulated the sensory distribution of an ulnar area of organic origin. A complete left hemihypesthesia and hemihypalgesia was also present. He complains of insomnia, headaches, partial loss of memory and general nervousness, with inability to stand excitement or hear noises "without jumping."

THE COLLOIDAL GOLD REACTION IN FOUR HUNDRED AND NINETY-EIGHT PSYCHIATRIC CASES *

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The following report is an analysis of the colloidal gold reaction in 498 cases of various psychoses in which there was suspected a nervous syphilis from the clinical findings of pupillary changes, exaggerated or lost reflexes, speech defects, histories of early cardiovascular involvement or apoplexies; with miscarriages, still births, defective mental development or more frank syphilitic manifestations in the immediate relations. No case was punctured which did not show either a positive Wassermann reaction of the serum with suggestive neurological symptoms, or neurological symptoms which made dependence on a negative serum inadvisable. The colloidal gold reaction is of special interest in regard to the reliability of the test as a means of diagnosis of syphilis when all other tests are negative and as a method of differentiation between general paralysis of the insane and cerebrospinal syphilis. Lange's original method was at first used, but later the modification of Miller and his colleagues of Phipp Psychiatric Clinic, Johns Hopkins, was substituted. No solution was employed which showed the faintest tinge of purple, brilliant orange-red solutions properly standardized being used. The Wassermann tests of serums and fluids were made by Dr. Sobei Ide in the laboratories of the Psychopathic Hospital, Ann Arbor, Mich. The spinal fluid was examined in the Kalamazoo State Hospital laboratories, the pleocytic count being a bedside count which we have found to be more reliable. The test for globulin is Nonne's Phase 1, checked up occasionally by the Ross-Jones modification. Other tests of the fluid were routinely made, but not recorded for the sake of brevity and clearness.

The disease types are grouped in order to facilitate conclusions in regard to the type of reaction which might be expected in definite disease entities, especially where neurological symptoms existed but a syphilitic infection was doubtful and yet impossible to rule out. A small number of the cases have been examined at necropsy and the nervous tissues worked up in the laboratories. They are mentioned in the group to which they belong.

* From the Laboratories of the Kalamazoo State Hospital, Michigan.

TABLE 1.—GENERAL PARESIS

Case Number	Wassermann Test		Cells	Globulin	Colloidal Gold
	Blood	Spinal Fluid			
15005	4	4	40	4	5555422100
15011	4	4	110	4	4443221000
15089	4	4	30	3	4444422200
15097	4	4	25	4	5555433210
15138	4	4	120	4	5555543200
15139	4	4	140	4	5555543811
15240	4	4	35	4	5555511110
15329	4	4	100	4	4444410000
15362	4	4	30	4	1112233100
15423	4	4	0	3	0002344110
15428	4	4	40	4	5555554432
15437	4	4	60	4	5555540000
15464	4	4	80	4	5544320000
15528	4	4	40	4	5544320000
15539	4	4	50	4	5544331100
15643	4	4	60	3	5544432000
15668	4	4	5	4	4443333200
15674	4	4	50	4	5555554300
15706	4	4	0	2	5554432000
15709	4	4	80	3	5554321000
15716	4	4	60	2	0023300000
15785	4	4	110	4	5544310000
15746	4	4	30	4	5544332200
15772	4	4	180	4	5533321000
15790	4	4	25	4	5555554300
15821	4	4	90	4	5555554321
15827	4	4	80	4	5555433210
16122	4	4	20	4	5555432000
16169	4	4	10	3	5555554320
16187	4	4	70	4	5555555310
16188	4	4	70	4	5555555410
16249	4	4	70	4	5555432000
16282	4	4	140	4	0112332100
16328	4	4	120	4	5544321000
16332	4	4	50	4	5555432100
16334	4	4	60	4	5555433321
16341	4	4	50	2	5554321000
16347	4	4	70	4	5553321000
16396	4	4	150	4	5555443210
16407	4	4	40	4	5555554320
16418	4	4	110	3	5554431000
16484	4	4	40	4	5555421000
16592	4	4	15	4	5555443200
16607	4	4	80	4	5555532100
16671	4	4	5	4	5554321000
16711	4	4	190	4	5554443211
16743	4	4	30	4	5555554210
16749	4	4	20	4	5555552100
16700	4	4	30	4	5555542100
16803	4	4	40	4	4444432110
16830	4	4	70	4	5555432100
16853	4	4	90	4	5555542110
16864	4	4	100	4	5555532100
16896	4	4	30	4	5554321000
14938	4	4	190	4	5555420000
15196	4	4	140	2	4555440000
16278	4	4	750	4	5555543320
16239	4	4	50	4	2343211000
14996	4	4	60	3	4422211000
15847	4	4	30	4	5555433100
16856	4	4	50	2	0123321000
15378	4	4	15	2	443322100
15396	4	4	50	3	324432100
15712	4	4	20	2	5544330000
15778	4	4	50	4	5555554210
15800	4	4	30	3	5555322000
17010	4	4	10	4	5555543200
16154	4	4	110	4	5544320000
16291	4	4	40	4	5554332000
16467	4	4	80	3	5543321000
16667	4	4	8	4	554331000
16725	4	4	60	4	5544331000
14397	4	4	10	2	2221000000
16830	4	4	10	3	5554332100
16940	4	4	80	4	5555544331
17034	4	4	10	4	5555432100
16968	4	4	30	4	5555432110
15121	2	4	40	4	5555532111

TABLE 1.—GENERAL PARESIS—Continued

Case Number	Wassermann Test		Cells	Globulin	Colloidal Gold
	Blood	Spinal Fluid			
15131	4	3	40	4	4444320000
15228	3	4	30	4	5555444430
15268	1	4	60	4	2234110000
15303	4	3	20	1	1111222210
15348	±	4	40	4	5554444000
15450	2	4	50	4	5554320000
15492	4	—	180	4	1111223111
15534	4	—	70	4	000033210
15602	3	4	20	3	555432100
15602	1	4	50	3	555443211
15752	3	4	170	4	555555200
15774	±	4	20	4	555552100
16274	4	—	90	4	0112222100
16417	±	4	50	4	555543100
16494	±	4	70	3	5544322100
16514	—	4	15	1	445552100
16576	±	4	60	3	5433210000
16618	3	4	5	4	555554320
16764	2	4	60	4	5555542110
16802	±	4	60	4	455552100
16825	±	4	40	4	5555553210
16235	±	4	20	4	5555543100
16280	4	2	10	4	5555553200
16376	4	—	10	1	5543200000
16397	3	4	50	4	555554320
16706	±	4	?	4	4444321100
15112	4	4	80	3	2233110000
15231	4	4	20	4	344443200
15262	4	4	80	4	4442221000
15314	1	4	20	4	1122221000
15426	4	4	50	3	5544332100
15429	±	4	30	3	4443320000
15434	4	4	220	4	111223321
15448	4	4	30	3	1123331000
16162	4	3	420	4	0011233210
16216	1	4	80	3	1232100000
16023	3	4	50	4	5544332100
17033	±	4	10	4	5543321000
16970	1	4	40	4	5555544331
17014	±	4	15	3	5554332100
16970	4	4	15	4	5555543210
15303	±	4	0	3	0000000000

Analyzing the general paresis group in which most consistent results have been obtained by various investigators, it is observed that, in the 120 cases examined, 96 gave typical parietic curves, of whom 78 showed correspondingly strongly positive Wassermann reactions of serums and spinal fluids with markedly positive globulin and pleocyte count. In the remaining eighteen cases there were variations in the Wassermann reactions and cell counts. Five cases gave a slightly atypical parietic curve, the reduction in the first one or two tubes being 4, then rising to 5 and following the normal curve; subsequent tests of similarly reacting fluids have frequently given typical curves, the variations being unaccounted for, and it has been our custom to consider them parietic, moreover the curves have been confirmed by necropsy. Thirteen cases gave syphilitic curves, four of which—Cases 15382, 16955, 15303 and 15492—received treatment, and six—Cases 15112, 15314, 15434, 15448, 16162 and 16216—were tabetic in type. Five cases gave atypical curves, among these Cases 15423, 14379 and 15534 had received treatment. One case which

had received treatment gave a practically negative reaction. Eight cases — 14936, 15112, 15774, 16278, 16334, 17010, 15196 and 15235 — came to necropsy and the diagnosis confirmed, Case 15112 being tabetic in type.

TABLE 2.—CEREBROSPINAL SYPHILIS

Case Number	Wassermann Test		Cells	Globulin	Colloidal Gold
	Blood	Spinal Fluid			
14831	—	—	20	2	2233220000
14516	±	4	100	2	0112322000
14546	4	4	210	4	1123330000
14806	4	4	80	3	1123110000
14971	4	4	40	3	0122210000
15087	4	4	10	2	1133221000
15099	—	—	6	1	1112332100
15100	—	±	0	—	1122210000
15310	4	4	50	4	2334443310
15332	4	4	50	4	1134411000
15333	2	4	20	3	1223332000
15414	4	4	350	2	1123321100
15467	4	4	20	4	1222233320
15622	4	4	30	4	0011221000
15672	—	—	50	2	0011233220
15680	4	3	10	1	0122100000
15721	3	—	20	3	0123310000
15734	4	4	30	1	2233320000
15764	—	—	0	1	1112221000
15780	4	—	0	0	0233211000
16033	—	—	5	1	1123320000
16067	—	4	30	4	0111232210
16178	4	4	20	4	4533332100
16280	—	±	0	2	0123210000
16335	—	4	30	3	0012321000
16365	—	—	10	1	0012210000
16370	4	—	3	2	0000000000
16390	—	4	0	2	0012321000
16394	4	4	310	2	0012321000
16425	4	±	20	2	1233110000
16443	—	—	3	—	0000000000
16490	—	—	5	2	0122100000
16461	4	4	50	2	1123321000
16512	—	—	0	1	0000000000
16529	2	—	0	—	0133100000
16564	4	4	110	4	0112333210
16586	—	—	15	3	1122211000
16582	—	—	0	1	0122100000
16609	4	4	40	4	1123321000
16658	2	4	40	4	0112332100
16665	—	—	10	1	0122210000
16689	4	4	20	3	0123210000
16707	—	—	10	4	1122110000
16710	—	—	2	—	0121000000
16733	4	—	?	—	0122100000
16742	4	4	120	4	1123321000
15470	3	2	50	2	0012331100
16815	—	—	10	1	0122100000
16823	4	1	10	2	1233200000
16867	—	—	0	2	0122100000
16968	3	—	0	—	0122100000
17029	—	—	5	1	0012100000
17017	—	—	0	3	0112110000
16922	2	2	3	1	0012110000
16951	—	—	10	3	0123321000
16954	4	4	210	4	0124321000
16992	—	—	0	1	0122100000
15711	—	—	3	1	0012210000
16523	—	—	10	1	0012210000

Among the fifty-nine cases of cerebrospinal syphilis, twenty-three gave syphilitic curves checked up by positive Wassermann reactions of both serums and fluids. Eleven cases gave syphilitic curves, eight of whom showed positive serums and negative spinal fluids and three positive fluids and negative serums. Twenty-three gave syphilitic

TABLE 3.—ARTERIOSCLEROTIC INSANITY

Case Number	Wassermann Test		Cells	Globulin	Colloidal Gold
	Blood	Spinal Fluid			
14619	—	—	0	—	0000000000
14954	—	—	0	2	0000000000
15056	—	—	0	—	0000000000
15057	—	—	0	—	0000000000
15062	—	—	0	1	0000000000
15122	—	—	0	1	2332110000
15122	—	—	0	1	0000000000
15157	—	—	0	2	0000000000
15190	—	—	0	1	1112110000
15202	—	—	0	1	0000000000
15214	2	—	0	—	0000000000
15255	—	—	12	1	0000000000
15315	—	—	5	1	0000000000
15326	—	—	0	—	0000000000
15365	—	—	0	—	0000000000
15395	—	—	0	—	0000000000
15589	—	—	5	3	1122210000
15616	—	—	40	2	0000000000
15626	—	—	0	1	0000000000
15628	—	—	0	1	0000000000
15724	—	—	0	1	0000000000
15726	—	—	0	1	0000000000
15804	—	—	0	—	0112210000
15809	—	—	0	1	0112321000
15822	—	—	5	1	0000000000
16103	—	—	3	—	0000000000
16115	—	—	3	—	0011221000
16128	—	—	80	1	0000000000
16134	—	—	10	—	0000000000
16172	—	—	0	1	0012100000
16189	—	—	0	1	0000000000
16199	—	—	5	—	0000000000
16200	—	—	10	3	0123210000
16206	—	—	5	3	0123210000
16222	—	—	5	3	0012210000
16223	—	—	3	3	0012210000
16227	—	±	5	2	0012221100
16228	—	—	5	2	0011211000
16246	—	—	3	4	0002332210
16248	—	—	20	4	0000000000
16279	—	—	0	—	0000000000
16298	—	±	0	—	0000000000
16306	—	—	40	4	0011223200
16318	—	—	0	1	0000000000
16361	—	—	10	—	0000000000
16396	—	—	0	1	0012210000
16412	—	—	5	1	0012211000
16432	—	—	2	1	0000000000
16462	—	—	0	1	0000000000
16500	—	—	0	—	0000000000
16507	—	—	0	4	0000000000
16509	—	—	0	1	0122210000
16516	—	—	0	1	0000000000
16518	—	—	15	—	0000000000
16522	—	—	10	2	0012110000
16544	—	—	10	1	0000000000
16551	—	—	3	1	0000000000
16572	—	—	0	—	0122100000
16591	—	—	15	1	0000000000
16601	—	—	0	3	0000000000
16608	—	—	0	2	0122100000
16613	—	—	10	—	0000000000
16615	—	—	10	—	0000000000
16659	—	—	0	1	0012100000
16669	—	—	5	1	0122100000
16674	—	—	5	—	0000000000
16709	—	—	8	4	0000000000
16712	—	—	20	—	0000000000
16714	—	—	10	1	0122100000
16722	—	—	20	—	0000000000
16730	—	±	0	—	0000000000
16737	—	4	0	4	0000000000
16774	—	—	0	1	0122100000
16784	—	—	0	2	0000000000
16792	—	—	10	1	0000000000
16807	1	—	0	—	0122100000
16870	—	—	10	1	0123210000

TABLE 3.—ARTERIOSCLEROTIC INSANITY—Continued

Case Number	Wassermann Test		Cells	Globulin	Colloidal Gold
	Blood	Spinal Fluid			
16875	—	—	5	1	000000000
16894	—	—	10	—	000000000
16909	—	—	0	—	000000000
16934	—	—	8	1	0122210000
16957	—	—	10	1	0012110000
16763	—	—	20	1	000000000
16943	—	—	0	—	000000000
16947	—	—	0	—	000000000

curves with negative Wassermann reactions of both serums and spinal fluids; clinically, these cases were largely arteriosclerotic in type with sluggish or absent pupillary reflexes, diminished or absent knee jerks, histories of early vascular involvement, symptoms of visceral syphilis, frequently a history of infection or of abortions and stillbirths in the wives, the cases being classed in the syphilitic group more on the clinical histories than on the syphilitic curve. Five of these—Cases 16365, 16460, 14831, 15711 and 16523—came to necropsy and showed, pathologically, well-marked evidence of an old endarteritis; syphilitic in type, of the brain vessels with evidence of visceral syphilis. Two cases gave negative reactions with negative serums and fluids, being placed in this class entirely on their clinical histories. The cell counts in seven cases were 100 and over, the average count being rather low for this form of syphilis though this may be due to the fact that the endarteritic type of the disease largely predominated. The acellular form of cerebral syphilis occurred chiefly among those having negative Wassermann reactions.

The arteriosclerotic group of eighty-five cases showed twenty-nine giving syphilitic curves with negative Wassermann reactions of the serums and spinal fluids. These cases lacked sufficiently convincing clinical histories pointing toward a syphilitic involvement to warrant classing them in the cerebral syphilis group on the gold curve alone. Fifty-six cases gave negative gold reactions, among whom one case gave a mildly positive Wassermann reaction of the serum and one a ++++ fluid. This latter case should have received another test. Cases 15202, 15255, 16189, 16875, 16507 and 15062 were given a necropsy examination, the first five confirming the gold curve. The last showed an old pathologic condition of the cerebral vessels highly suggestive of syphilis.

Among the forty-eight defectives, syphilitic curves were given in fourteen, negative reactions in thirty-three and paretic in one—the last showing fifteen cells, ++++ globulin reaction and negative Wassermann reactions of serum and spinal fluid. The group was

composed of morons, imbeciles and idiots with epilepsy, periods of disturbances, hysterical episodes or drug addiction. In Case 16339 the patient was suffering with alcoholic hallucinosis. The positive syphilitic curves in this series are of special interest from the fact that a fairly large number of the defectives in the institution in whom an acquired syphilis could with a moderate degree of certainty be ruled

TABLE 4.—DEFECTIVES

Case Number	Wassermann Test		Cells	Globulin	Colloidal Gold
	Blood	Spinal Fluid			
14638	—	—	5	—	0000000000
15028	—	—	0	—	0000000000
15033	—	—	0	—	0000000000
15048	—	—	b	1	0000000000
15213	1	—	5	2	1122100000
15285	—	—	0	—	0000000000
15305	—	2	0	—	0000000000
15372	—	—	5	2	0000000000
15423	—	—	5	—	0000000000
15494	—	—	0	2	0000000000
15514	—	—	0	—	0000000000
15648	—	—	10	—	0000000000
15658	—	—	5	—	0000000000
15745	—	—	0	1	0000000000
15787	—	—	c	—	0000000000
15793	—	—	0	—	0000000000
15816	—	—	0	—	0000000000
15932	—	±	30	1	1122210000
16139	—	—	0	—	0000000000
16193	—	—	0	—	0000000000
16305	—	—	5	1	0012110000
16313	—	—	5	1	0012110000
16330	—	—	5	4	0000000000
16374	—	—	0	—	0000000000
16398	—	—	0	—	0000000000
16399	—	—	0	—	0000000000
16400	—	—	10	1	0012110000
16457	4	—	0	1	0112110000
16583	—	—	40	—	0000000000
16603	—	—	10	2	0012210000
16692	—	—	2	1	1121100000
16731	—	—	3	—	0000000000
16171	—	—	15	4	5555532000
16192	—	—	10	2	0000000000
16228	—	—	0	—	0000000000
16323	—	—	10	—	0000000000
16333	—	—	5	1	0112110000
16350	—	—	8	—	0000000000
16753	—	—	0	—	0000000000
16772	—	—	0	—	0000000000
16805	—	—	10	2	0122110000
16617	—	—	0	1	0122210000
16672	—	—	5	—	0000000000
16634	—	—	0	1	0122100000
16690	1	—	0	—	0012110000
16692	—	—	0	—	0000000000
16925	—	—	0	3	0122110000
16296	—	—	0	0	0000000000

out have shown the physical stigmata of a syphilitic germ infection rather than a germ damage as seen in the diminished or lost reflexes, cerebral palsies, hydrocephalus, etc., and yet have uniformly given negative Wassermann reactions of the serums and spinal fluids. In a number of these cases there have been demonstrated at necropsy evidences of cerebral endarteritis, diffuse sclerosis, fibrous meningitis and pachymeningitis, porencephaly, etc., suggestive of a syphilitic

etiology, and we have been forced to draw the conclusion that after the active process dies out there is an increasing tendency in the later adult years to a progressive disappearance of the antibodies in the body fluids with negative reactions. It is therefore possible that the syphilitic reactions obtained in these cases may be of diagnostic value on account of the extreme sensitiveness and apparent specificity of the gold solution. Unfortunately, none of the present series of defectives came to necropsy and those on whom the above conclusions were drawn were not subjected to the gold test.

TABLE 5.—MANIAC-DEPRESSIVE PSYCHOSIS

Case Number	Wassermann Test		Cells	Globulin	Colloidal Gold
	Blood	Spinal Fluid			
14063	±	—	3	4	0112100000
14229	—	?	40	4	1112224400
14567	±	—	10	—	3321100000
14867	4	—	15	—	0000000000
15028	—	—	20	—	0000000000
15122	1	—	0	—	2221110000
15207	—	—	8	—	0000000000
15267	4	—	5	—	0000000000
15267	—	—	5	2	4433310000
15292	4	—	0	2	0000000000
15309	1	—	0	1	0000000000
15336	—	—	20	2	1112223330
15378	—	—	5	1	0000000000
15407	—	—	160	2	0123332000
15430	—	—	0	—	0000000000
15639	—	—	0	—	0000000000
15701	±	—	5	—	0000000000
15718	—	—	0	—	0000000000
15725	—	—	0	—	0000000000
15747	±	—	0	—	0000000000
15755	—	—	0	1	0012000000
16076	—	1	4	1	0000000000
16100	—	—	20	4	0012211000
16119	—	—	3	—	1112210000
16128	—	—	0	2	0011210000
16259	—	—	10	1	0012210000
16303	—	—	0	—	0122100000
16324	—	—	0	—	0000000000
16467	3	—	0	—	0000000000
16535	—	?	?	—	0000000000
16573	—	—	5	2	0000000000
16586	—	—	10	—	0000000000
16626	—	—	0	—	0000000000
16682	4	—	10	4	0122100000
16686	—	—	5	1	0000000000
16687	—	—	3	1	0000000000
16702	—	—	0	3	0122210000
16777	—	—	0	—	0000000000
16781	—	—	0	1	0000000000
16924	—	—	20	1	0000000000
17031	—	—	0	1	0000000000
16831	—	2	30	2	0123210000
16916	—	—	0	1	0000000000

The manic-depressive group comprised forty-three cases. An attempt was made in these cases to rule out a symptomatic mania or depression due to a syphilitic infection the cases being considered circular insanity with concomitant syphilis and grouped accordingly. Among the forty-three, twelve gave syphilitic curves, twenty-eight negative reactions and three suggestive parietic curves. Four cases gave positive Wassermann reactions of the serums with negative spinal

fluid reactions in three and a syphilis curve in one. Only one spinal fluid gave a positive Wassermann reaction. Twelve cases gave a pleocytosis above normal.

TABLE 6.—EPILEPTICS

Case Number	Wassermann Test		Cells	Globulin	Colloidal Gold
	Blood	Spinal Fluid			
14354	—	—	0	—	000000000
14902	—	—	40	4	111283711
15051	—	—	0	—	000000000
15184	—	—	3	—	000000000
15281	—	—	0	—	000000000
15381	—	—	3	—	000000000
15362	—	—	0	—	000000000
15391	—	—	0	3	000000000
15393	—	—	0	—	000000000
15398	—	—	0	—	000000000
15468	—	—	20	2	000000000
15505	—	—	10	2	222110000
15516	—	—	0	1	000000000
15536	±	—	5	1	000000000
15698	—	?	0	1	000000000
15756	—	—	10	1	112211000
15759	—	—	0	1	001221100
15766	—	—	5	—	112211000
15805	—	—	0	—	000000000
15820	—	—	0	—	000000000
15824	—	—	3	—	000000000
15840	—	—	0	—	000000000
16099	—	—	0	—	000000000
16206	—	—	0	—	000000000
16231	—	—	0	1	001210000
16255	—	—	0	—	000000000
16306	—	—	0	2	000000000
16351	—	—	3	1	000000000
16362	—	—	0	—	000000000
16366	—	—	0	—	000000000
16475	—	—	20	4	000121100
16478	—	—	0	—	000000000
16515	—	—	2	—	000000000
16562	—	—	5	—	112210000
16660	4	—	5	1	000000000
16685	—	—	0	2	012210000
16727	—	—	5	2	001210000
16855	—	—	5	—	012210000
16971	—	—	10	4	111128821
17013	—	—	0	3	001221000

There were forty cases in the epileptic group, twelve giving syphilitic curves, twenty-seven negative reactions and one a suggestive incipient parietic curve. One case showed a positive Wassermann reaction of the serum with a negative gold curve. All spinal fluids gave negative Wassermann reactions. Six cases showed a cell count above normal.

The twenty-eight cases in the dementia praecox group formed one of the most interesting. Nine syphilitic curves were given, two in the hebephrenic and seven in the paranoid group. Eighteen cases gave no curves. Case 15760 was positive in all the reactions, being diagnosed a praecox with cerebral syphilis, alcoholic paranoia not excluded. Seven cases showed a cell count above normal. Cases 15815 and 16104 came to necropsy, the first demonstrating no evidence of syphilis, the second showing a very moderate endarteritis of the basal brain vessels.

TABLE 7.—DEMENTIA PRAECOX

Case Number	Wassermann Test		Cells	Globulin	Colloidal Gold
	Blood	Spinal Fluid			
15681	—	—	5	—	0000000000
16411	—	—	0	1	0012210000
16482	—	—	0	—	0000000000
16639	—	—	3	—	0000000000
16821	4	—	20	2	0011211000
16861	—	—	0	—	0000000000
16862	—	—	0	—	0000000000
15074	—	—	0	—	0000000000
15018	—	—	0	1	0000000000
16717	—	—	0	—	0000000000
15049	—	—	0	—	0000000000
15191	3	—	0	—	0000000000
15471	—	—	2	—	0000000000
15689	—	—	0	1	0000000000
15703	—	—	0	—	0000000000
15760	4	4	10	2	2223320000
15834	±	—	15	1	0012211000
15815	—	—	10	—	0000000000
16203	—	—	0	3	0122100000
16221	—	—	5	1	0122100000
16303	—	—	0	1	0122100000
16342	—	—	0	—	0000000000
16504	—	—	2	1	0000000000
16653	—	—	5	—	0000000000
16700	—	—	80	3	0122110000
16849	—	—	0	—	0000000000
16893	—	—	15	1	0122100000
16104	—	—	10	—	0112210000

TABLE 8.—INEBRIATES

Case Number	Wassermann Test		Cells	Globulin	Colloidal Gold
	Blood	Spinal Fluid			
15006	4	—	10	2	0000000000
15047	±	—	10	1	0000000000
15208	—	—	20	—	0000000000
15204	±	—	0	—	0000000000
15297	—	—	0	1	0000000000
15299	—	—	7	3	0011122000
15408	—	—	0	—	0000000000
15507	—	—	0	3	0000000000
15513	—	—	0	2	0000000000
15582	—	—	0	—	0000000000
15634	4	—	5	1	0000000000
15684	—	—	0	1	0000000000
15710	—	—	0	—	0000000000
16068	—	4	10	—	0011220000
16129	4	—	0	1	0000000000
13976	4	±	0	—	0000000000
16240	—	—	0	—	0000000000
16252	—	—	0	—	0000000000
16281	—	—	10	1	0122110000
16287	—	—	5	1	0000000000
16429	—	—	3	1	0012110000
16463	4	—	6	1	0000000000
16485	—	—	10	1	0012110000
16567	—	—	15	3	0012100000
16695	—	±	30	1	0112210000
16761	—	—	5	—	0012110000
16760	—	4	0	1	0122100000
16456	—	—	0	1	0011210000
16862	—	—	50	—	0121000000
16820	—	±	2	—	0012110000

Among the thirty inebriates, twelve gave syphilitic curves and eighteen showed no curves. Cases 16761 and 16760 were paranoics, Case 16695 an hallucinosis and Case 15820 a delirium tremens.

TABLE 9.—DRUG HABITUÉS

Case Number	Wassermann Test		Cells	Globulin	Colloidal Gold
	Blood	Spinal Fluid			
14821	—	?	3	2	1112200000
15066	4	—	10	2	0011122100
15233	—	—	5	1	0000000000
15237	±	—	6	1	0000000000
15321	±	—	5	—	0000000000
15416	4	—	10	2	0011122100
15681	—	—	3	1	1122100000
16043	—	—	0	1	0000000000
16080	—	—	0	—	0000000000
16132	±	—	0	1	0012210000
16201	±	—	3	2	0012110000
16371	—	—	5	1	0012210000
16668	—	4	10	1	0122100000
16693	—	—	0	2	0122110000

Among the fourteen drug cases, nine gave syphilitic curves, three being reinforced by positive Wassermann reactions. In Case 16693 the patient was a tabetic.

TABLE 10.—MISCELLANEOUS GROUP

Case Number	Wassermann Test		Cell	Globulin	Colloidal Gold	
	Blood	Spinal Fluid				
15491	—	—	0	2	0000000000	Exhaustion psychosis
15655	—	—	2	—	0000000000	Exhaustion psychosis
16212	—	—	0	—	0000000000	Exhaustion psychosis
16222	—	—	8	—	0012210000	Exhaustion psychosis
16304	—	—	0	—	0000000000	Exhaustion psychosis
16496	—	—	3	—	0000000000	Exhaustion psychosis
16580	—	—	0	1	0000000000	Exhaustion psychosis
16755	—	—	400	1	0000000000	Exhaustion psychosis
16812	—	—	?	—	0000000000	Exhaustion psychosis
16880	—	—	0	—	0000000000	Exhaustion psychosis
16921	—	—	10	1	0122210000	Exhaustion psychosis
15021	—	—	15	2	0000000000	Huntington's chorea
15461	—	—	5	—	0000000000	Huntington's chorea
15638	—	—	5	—	0000000000	Huntington's chorea
15731	—	—	0	1	0000000000	Huntington's chorea
15812	—	—	0	—	0000000000	Huntington's chorea
16284	—	—	2	—	0000000000	Huntington's chorea
16853	—	—	2	—	0000000000	Huntington's chorea
15136	±	—	10	—	0000000000	Hysteria
15168	—	—	0	—	0000000000	Hysteria
16657	—	—	3	—	0121000000	Hysteria
15307	2	—	3	—	0000000000	Delirium (drugs)
15535	—	—	0	—	0000000000	Delirium (organic dementia)
15799	—	—	0	—	0012210000	Delirium (org. brain disease)
17016	—	—	0	—	0000000000	Paralysis agitans
15268	—	—	5	—	0000000000	Paralysis agitans
15268	±	—	8	2	1122220000	Psychopath
15807	—	—	220	4	1111221000	Senile (postmortem fluid)
16734	—	4	40	3	1122110000	Tabes; not insane
15166	—	—	15	1	1221110000	Brain tumor (sarcoma)
15052	—	—	0	—	0000000000	Ideational apraxia

In the miscellaneous group, in Case 16755, the specimen was a postmortem fluid which accounted for the marked pleocytosis. Case 15638 among the Huntington's chorea group was given a necropsy examination and revealed an old, rather moderate lumbar tabes with a very moderate endarteritis of the pial vessels. Case 15166 was examined at necropsy showing a sarcomatous tumor in the region of the thalami.

SUMMARY

The following results were obtained from the gold test on the fluid of the 498 cases: 103 gave paretic curves with positive Wassermann reactions of either serums or spinal fluids, or both; 10 gave suggestive curves of incipient paresis with negative Wassermann reactions of serums and spinal fluids; 53 gave syphilitic curves with positive Wassermann reactions of either serums or spinal fluids, or both; 118 gave syphilitic curves with negative Wassermann reactions of serums and spinal fluids; 209 gave negative gold reactions, 14 of whom showed positive Wassermann reactions of the serums and 3 positive Wassermann reactions of the spinal fluids; 5 gave atypical curves, several of these being treated cases.

CONCLUSIONS

1. The spinal fluid of cases of dementia paralytica causes a quite characteristic curve with the colloidal gold solution which is of such frequent occurrence as to be diagnostic. Seven of our cases were confirmed by necropsy.

2. The spinal fluid of taboparetics may normally give syphilitic curves or rather low paretic curves as evidenced by the reactions of a number of our paretics and as confirmed by necropsy.

3. Cerebrospinal syphilis gives a curve which may be considered diagnostic.

4. Syphilitic curves with negative Wassermann reactions of serums and spinal fluids may be obtained in cerebrospinal syphilis having been demonstrated pathologically in five of our cases, the reaction not necessarily being due to a dialyzable substance other than syphilitic.

5. The gold curve is of value in clearing up the etiology of old arteriosclerosis with negative Wassermann reactions; necropsies having demonstrated a syphilitic type of vascular lesion in cases giving syphilitic curves and a simple senile degeneration of the vessels in cases giving negative gold reactions.

6. It is of interest that five cases of Huntington's chorea gave negative gold reactions, one of which, however, demonstrated pathologically an inactive moderate tabetic involvement of the lumbar regions.

7. The syphilitic curve may be of value in clearing up the etiology of mental deficiencies after an active syphilitic process has ceased and antibodies have disappeared from the body fluids.

8. The globulin reaction is rather uniformly strongly positive in paretics and a fair proportion of syphilitics, but there appears to be no definite relationship between the curve and the strength of the reaction.

THE RÔLE OF THE PITUITARY GLAND IN EPILEPSY

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The problem of epilepsy may be best approached by considering all convulsions which are accompanied by unconsciousness, whether periodic or otherwise, as symptoms of some underlying pathologic state or disease. By doing this the convulsion becomes a symptom. The question then arises, which I believe should be answered in the negative, whether any so-called epileptic state should, of itself, be considered a disease.

ETIOLOGIC FACTORS

Thus, among the underlying causes of convulsions are uremia, eclampsia and other toxicoses; several varieties of trauma, as birth trauma and that from blows on the head; certain intracranial irritations, as brain tumors and cerebral hemorrhages; certain infections, as syphilis and varieties of meningitis; certain conditions of cerebral maldevelopment accompanied by a degree of brain cell deterioration; *and certain conditions of the pituitary gland causing a change in its secretion.*

I believe, therefore, that convulsions, whether periodical and termed epilepsy, or not, are due to definite pathologic cerebral tissue changes which may be congenital or induced, and hence, that epilepsy is an organic and not a functional condition. In the toxic and pituitary cases there may exist previous malformation and instability of the cortical cells, or, on the other hand, it is perfectly conceivable that the toxic substance or the changed pituitary secretion may produce their own cortical cell deterioration. I published a paper on the relation of the pituitary gland to some cases of epilepsy, and in 1914¹ and again in 1916,² a paper on the subject of hypopituitarism in its relation to epilepsy. These papers were the result of a personal suggestion of Dr. Harvey Cushing and also of the perusal of his most excellent book.³

* Read before the Section on Nervous and Mental Diseases at the Seventieth Annual Session of the American Medical Association, Atlantic City, N. J., June 11, 1919.

1. Tucker, B. R.: South. M. J. 7:608 (Aug.) 1914.

2. Tucker, B. R.: Virginia M. Semi-Month. 31:1 (April 7) 1916.

3. Cushing, Harvey: The Pituitary Body and Its Disorders, Philadelphia, W. B. Saunders Company.

Dr. Daniel D. Talley has roentgenographed most of my cases and in nearly all instances the roentgenographic findings have confirmed the clinical diagnosis of either the permanent or the transitional stage of hypopituitarism. Dr. Johnston of Pittsburgh, I understand, was one of the earliest roentgenographers in noting pituitary changes in certain cases of epilepsy. In a paper read⁴ before this section in 1918 I described transitory pituitary changes, causing psychoses occurring during adolescence. Some of these transitory changes appeared also to be the cause of convulsive states. Cushing believes that the secretion of the posterior lobe of the pituitary joins the cerebrospinal fluid and thus bathes the cerebral cortex with a substance which is necessary to the functional stability of the cortical cells. When this secretion is diminished or absent the cells may be said to deteriorate. Whether this deterioration is due to the lack of whatever the pituitary secretion may give them, or whether it takes place by the lack of this substance allowing toxic substances to exert a deleterious influence, I am not sure. At any rate it appears that the administration of pituitary gland extract tends to alleviate symptoms caused by undersecretion of the pituitary gland.

TWO TYPES OF CASES

There are two forms of hypopituitarism here described: one chronic or congenital, and the other transitional. In *chronic or congenital types*, the patients have, among other indications, increased fat, lack of body hair, feminine distribution of pubic hair in the male, scanty menstruation in the female, diminished perspiration, increased sugar tolerance, small genitalia, and frequently bradycardia and lowered blood pressure. In the *transitional type* the patient may show evidence of normal pituitary secretion, or hypersecretion in the past, which during adolescence becomes lessened. This is at times traceable to some illness or trauma. In these cases the patient begins to take on flesh, has an increased desire for sweets, become less energetic, eats voraciously, perspires less and may have slow pulse rate and lowered blood pressure. In either type occasional or periodic convulsions may occur, which usually make their appearance during adolescence. Convulsions may also occur in cases of tumor of the pituitary body, as pointed out by Cushing, but the discussion of tumor cases is not included in this paper.

PATHOLOGIC CHANGES OBSERVED

Roentgenographic examinations in the chronic or congenital type of hypopituitarism have shown small fossae with large clubbed pos-

4. Tucker, B. R.: Pituitary Disturbance in Its Relation to the Psychoses of Adolescence, J. A. M. A. 71:330 (Aug. 3) 1918.

terior processes and frequently elongated anterior processes tending to bridge the fossa. Many of these cases have shown irregularity in the contour of the posterior part of the sella.

In the transitory type roentgenographic examinations have revealed either normal-sized or enlarged fossae, according to whether the clinical symptoms have pointed to normal or hypersecretion in the past. The posterior processes have been enlarged and clubbed. The anterior process may be enlarged and, at times, bridging between the processes has been observed. Roughening of the posterior contour of the sella is usually found.

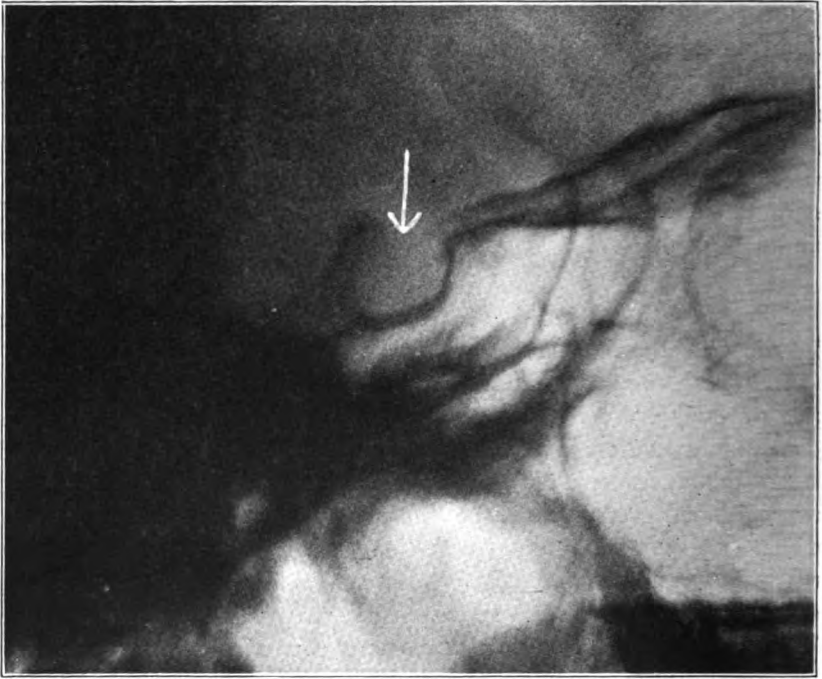


Fig. 1.—Large fossa, elongated processes slightly roughened; hyperpituitarism.

The roentgenographic difference, therefore, between the two types lies chiefly in the size of the fossae, that of the chronic type being decreased and that of the transitional type being normal or enlarged in general contour even if encroached on by bony growth.

AUTHOR'S OBSERVATIONS

The present investigation is based only on the study of my last 200 cases of epilepsy, for the reason that I do not feel that previous to these cases I had knowledge or experience enough in the pituitary

type of convulsions to examine them sufficiently or judge of them accurately.

Of the 200 cases, sixty-three, or 31.5 per cent., revealed some evidence of pituitary disturbance. Of these sixty-three cases, thirty-five were discarded for the reason that they had in addition to clinical and roentgenographic signs of dispituitarism, evidence of syphilis, marked nephritis, active toxemia from focal infections, hydrocephalus,

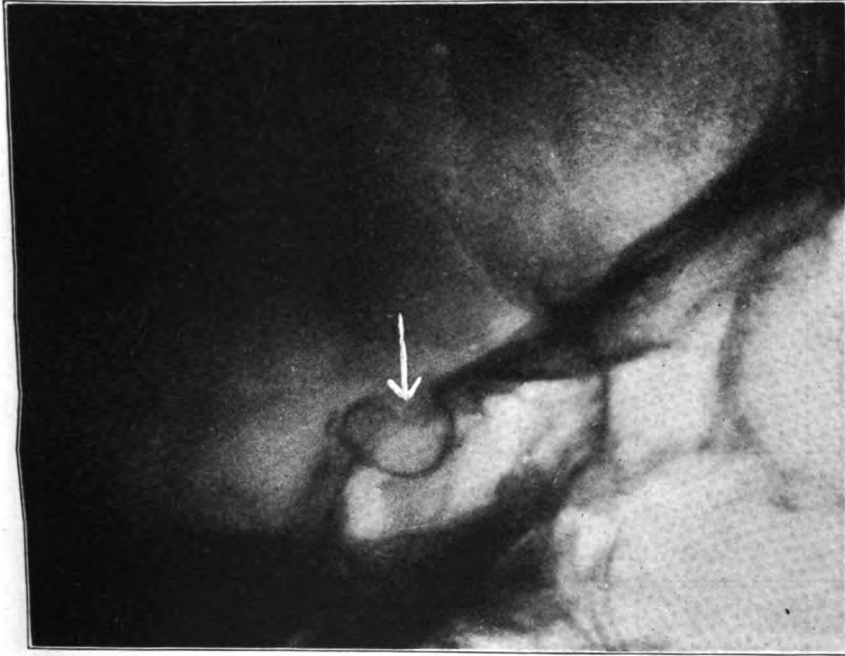


Fig. 2.—Small fossa, anterior and posterior processes approximated; hypopituitarism.

organic brain disease or injury, or because the evidence of pituitary disturbance was so slight that it was negligible. However, in most of these, pituitary feeding was beneficial. We thus have left *twenty-eight pure pituitary cases, or 14 per cent.* Of these twenty-eight cases, seventeen were of the chronic hypopituitary type, and eleven of the transitional hypopituitary type.

TREATMENT

In the treatment of these cases if bromid was being taken small doses were continued, and in some of the severe cases small doses were given, especially at first. The effort, however, was made to get the patient off of bromid.

TABLE 1.—CHRONIC HYPOPHYSECTOMY TYPE *

Case	Age of Onset of Attack	Type of Attack	Average Frequency of Grand Mal	History of Injury or Disease	Preparation of Gland Used	Result of Feeding	Remarks
1	11	Grand	1 a month	Injury to head when 7 years old	Whole gland	Two in 18 months	Marked improvement
2	14	Grand	1 every month	None	Whole gland	None in 3 years	Cured
3	18	Grand	1 every 6 weeks	Head injury as child	Whole gland	None for 14 months	Attacks ceased
4	18	Grand	1 every 2 months	None	Anterior lobe gland	1 every 2 months	Not improved
5	11	Grand	1 a month	None	Whole gland	Three in 18 months	Marked improvement
6	12	Grand	2 to 3 a week	None	Whole gland	No attacks over 3 years	Cured
7	10	Petit	No grand, petit daily	Injury to head as child	Whole gland	No attacks over 3 years	Cured
8	13	Petit	No grand, petit many a day	None	Whole gland	None for over 3 years	Cured
9	12	Grand	3 a year	None	Whole gland	None for over a year	Prospects of a cure
10	13	Grand and petit	1 to 2 a week	Head injury as a child	Whole gland	One a month	Improvement
11	9	Grand	3 or 4 a year	Head injury 5 years old	Anterior lobe gland	None for 2 years	Prospects of cure
12	30	Grand	1 every 6 weeks	None	Whole gland	One in 2 years	Prospects of cure
13	17	Grand	3 a year	Malaria	Whole gland	Same as before	Took medicine irregularly
14	15	Grand	1 a week	None	Whole gland	One in 2 months	Marked improvement
15	13	Grand	1 every 6 weeks	Rheumatism	Whole gland	One every 3 months	Improvement
16	11	Grand	1 a month	None	Whole gland	None for over 2 years	Apparent cure
17	8	Grand	1 every month or 2	None	Whole gland	None for 6 months	Marked improvement

* All of these patients had definite evidence of clinical hypopituitarism and roentgenographic signs of the chronic type of hypopituitarism. The general health and mental condition of the patients improved while taking pituitary extract.

TABLE 2.—TRANSITIONAL HYPOPHYSECTOMY TYPE *

Case	Age of Onset of Attack	Type of Attack	Average Frequency of Grand Mal	History of Injury or Disease	Preparation of Gland Used	Result of Feeding	Remarks
1	18	Grand	1 every 2 months	None	Anterior lobe gland	One attack 18 months	Marked improvement
2	25	Grand and petit	1 every 2 months	None	Whole gland	Recent	Too recent to judge
3	13	Grand	1 every 3 weeks	Injury to head as child	Whole gland	Two in 9 months	Marked improvement
4	24	Grand	1 or 2 a month	None	Whole gland	Same as before	No improvement
5	20	Grand	1 every 6 months	None	Anterior lobe gland	None for 2 years	Prospects of cure
6	12	Grand and petit	1 a week	None	Whole gland	None for 18 months	Prospects of cure
7	17	Grand	1 every 2 or 3 months	Injury to head as child	Whole gland	One series of several attacks	Improvement
8	17	Grand	2 to 4 a week	None	Whole gland	No attacks for 8 mo.	Prospects of cure
9	22	Grand	1 a month	None	Whole gland	One every 2 months	Improvement
10	22	Grand and petit	3 a week	Injury to head few years before	Whole gland	None for 18 months	Prospects of cure, petit attacks rare
11	13	Grand	1 a week	None	Anterior lobe gland	One every 2 months	Marked improvement

* All of these patients had definite evidence of clinical hypopituitarism and roentgenographic pictures revealed the transitional type of that condition. It was noted that the general health and the mental condition of the patients greatly improved in all cases.

CONCLUSION

I believe that there is a definite relation between the undersecretion of the pituitary gland and a group of periodic convulsive attacks usually termed epilepsy; that this group is divided into a chronic hypopituitary type and a transitional hypopituitary type by both clinical and roentgenographic evidence; and that pituitary gland feeding has a markedly beneficial effect not infrequently leading to cure.

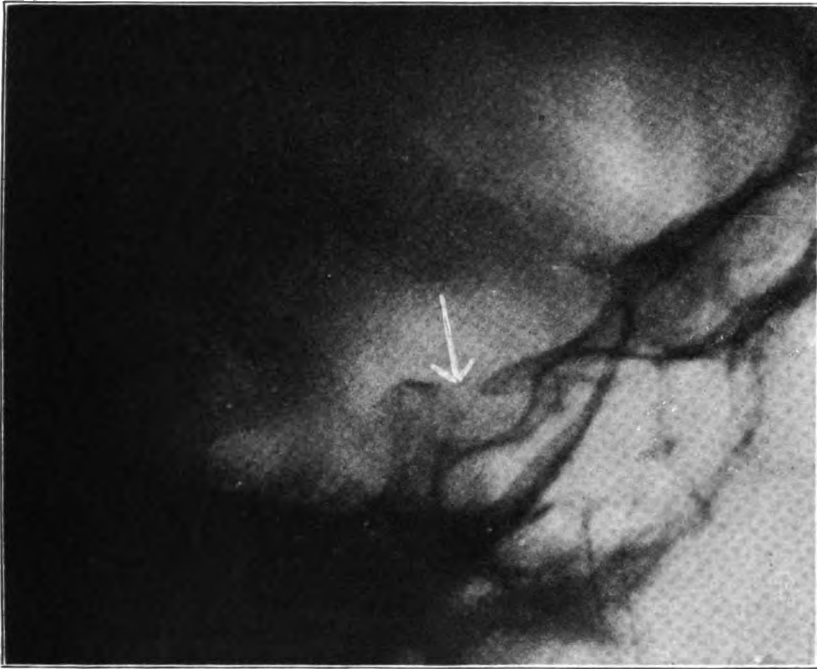


Fig. 3.—Moderate-sized fossa, enlarged roughened processes; transitional case.

DISCUSSION

DR. WALTER TIMME, New York: Dr. Tucker's report on what he has done is perhaps really too modest. The number of phases we see of this type that can be helped as far as the epilepsy is concerned, is quite large. The cures are cures as effective and quite as prompt as he has suggested. It remains to determine which are the proper cases for such therapy. You cannot from the appearance of the sella turcica on the roentgenogram determine if the person has epilepsy. Every hypopituitary case in which the patient has epilepsy shows sella changes, but every case with sella changes does not necessarily mean that the individual has epilepsy. You must remember also that the formation of the sella turcica does not necessarily go hand in hand with equivalent changes in the hypophysis. If the sella is large you can have all kinds of changes in the hypophysis without any effect on the sella. Sir Victor Horsley made an interesting experiment in this direction. He found the cerebral cortex in dogs was

more sensitive to electric currents after removal of the hypophysis than before. That led him to suspect that the irritability of the cortical cells was in a measure due to the lack of secretions of the hypophysis. As a matter of fact, these hypopituitary individuals, even though apparently sluggish, have also increased cortical irritability. They improve by feeding on pituitary extract. In Plattsburg we saw a man who had epileptic convulsions and the case was recognized as one of dyspituitarism of the Froelich dystrophia type. He was put on pituitary treatment and thereafter had only one convulsion in six months, previously having had convulsions daily, or three or four times a week. I have two slides which, with the permission of the chairman, I will show, demonstrating this phase of the situation. They are both cases of dyspituitarism, but show absolutely different physical signs. The acromegalic had epilepsy, the Froelich dystrophia type had not. The acromegalic was cured by administration of pituitary substance.

DR. H. H. HOPPE, Cincinnati: I have been struck with the relation between certain types of dyspituitarism and the appearance of epilepsy, especially the type which shows changes of a sexual character in young individuals. I had one case in particular, a boy who had feminine characteristics and large masses of fatty distribution. He has been under treatment four or five years with extract of pituitary gland, and there has been no change in the number of attacks or in the character of the epileptic attacks as a result of the administration of pituitary gland. Perhaps I made the mistake of limiting him to the anterior lobe. The anterior lobe stands in close relation with the development of sexual characteristics and if you have disease of the anterior lobe you will have heterogeneous sexual characteristics. This boy showed typical feminine characteristics, and the change that has occurred in him has been the development of characteristics of a masculine nature. He has grown to be a strong, slender, handsome man, all the feminine characteristics have disappeared except the masses of fat, but the epileptic attacks have not been changed by the administration of the anterior portion of the gland. I consider it was the dyspituitarism which gave rise to this case and to the abnormal sexual development. I have another case in which there has been no result from treatment, perhaps on account of the same limitation to the anterior lobe.

DR. E. B. ANGELL, Rochester, N. Y.: I wish to corroborate the statement made by Dr. Tucker and assure Dr. Hoppe that if he would use the extract of the whole gland he would get results. I believe it is the cases that occur during puberty that receive the most benefit from this therapy. I have been following the same process in a mild form of dementia praecox and have obtained similar results. I have had several remarkable results with extract of the whole gland in several cases of dementia praecox with the very type that presents some of these symptoms at puberty.

DR. GEORGE A. MOLEEN, Denver: I believe we are wise to believe that epilepsy and dementia praecox are produced by the same conditions. I think we all realize that when we find a disproportional secretion of the endocrine glands we are entitled to consider this as due to disequilibrium in the same fashion as the hormones are considered. I do not see why we cannot associate a form of epilepsy to the hormonal inequilibrium any more than we can associate epileptic seizures with alterations in the sella turcica. We find many patients with pituitary evidences predominating, but more are without epilepsy than with. That is particularly true in the normal type of pre-

adolescent deficiency. We are able to interpret them as the result of hormonal disequilibrium rather than as disease of one of the structures, namely, the pituitary itself.

DR. ALBERT E. STERNE, Indianapolis: Some years ago I used the anterior lobe of the pituitary in a large number of cases of epilepsy and had no results whatever. I came to the conclusion that it was absolutely ineffective and have seen no reason to change that opinion since. I have had no beneficial results whatever in the administration of the anterior lobe of the pituitary. Dr. Timme said yesterday that the anterior lobe extract was rather dangerous. My experience has been quite the contrary. He stated that in using the whole pituitary he used fresh substance. I would like to know where he obtains the fresh gland. We have had difficulty in getting the substance of any of the gland. The general results with feeding of the whole substance formerly was appreciably better than with the anterior or posterior lobes alone, but I cannot say that our results in several hundred epileptics have been anything like as pronounced as the report of the essayist, Dr. Timme and others. Either the substance they used is different or else they have had better success in the administration. There is another point I would like to mention, in reference to the roentgenograms of the sella turcica that were shown by Dr. Timme. They were all taken from the lateral point of view. Recently we have been taking our roentgenograms from the top directly so the rays enter through the infundibular opening. That gives us the dimensions from side to side. We have found that the sella turcica thus shows up better. We sometimes think we are dealing with an imprisoned hypophysis when we are merely judging from a side view.

DR. WALTER TIMME, New York: With the permission of the chairman I will answer the question regarding the extract of the pituitary substance. I usually have a man go to the abattoir and get the whole substance. He dries it and puts it in capsules. Occasionally we use the steer, but usually the hog. The Armour preparation is fairly fresh, and if taken within a reasonable length of time and kept cold and dry it will answer. Regarding the roentgenograms I showed, they were taken almost directly laterally and always stereoscopically and one directly down, but only one picture was shown on the screen which gave the general structural outline to the best advantage.

DR. IRVING J. SANDS, Brooklyn: In regard to the roentgenographic changes in the sella turcica I have tried to check them up at necropsy in fifty cases and I have not found any relation between the pituitary gland and the size or shape of the sella. We have had roentgenograms made of a few of these patients antemortem, and there was no relation between the changes in the pituitary and the sella turcica. I believe that with a few exceptions, such as acromegaly, the sella has no more to do with the condition or function of the pituitary gland than the shape of the nose has to do with one's respiration. I believe that the fibrous condition of the capsule has more to do with the functional possibilities of the pituitary gland because we have found many changes in the capsule where there were none in the sella itself.

DR. BEVERLEY R. TUCKER, Richmond, Va.: I think the shape of the nose has a good deal to do with the respiration. As far as the glandular substance is concerned, there are few patients to whom I give the anterior lobe, particularly if the blood pressure happens to be high. I give the whole gland capsule. There are three elements that must be present in these cases. If you want to have results you have to have the age of adolescence, the clinical type

and the roentgenographic findings. It is very hard to get a necropsy on an epileptic, but I would not take the finding on a case that did not have convulsion as a criterion at all. There are two distinct types of chronic hypopituitary cases. It is usually after puberty that these convulsions begin. Some give a history of having been normal until receiving a blow on the head or suffering from an acute infection after which the convulsions began. It seems to stir up something in the gland.

I remember ten or twelve years ago a neurologist made the statement before our Section that there was no such disease as neurasthenia. We were shocked then, but I think we have all come around to his opinion today. We may as well assume now there is no such disease as epilepsy which is always due to some distinct pathologic condition, and we will thereby separate off one group after another and treat the convulsions as a symptom, and cure the condition incidentally. Looking over these 200 cases, I found that ten were syphilitic; they had positive Wassermann reactions. These people should not be considered epileptics; they should be considered syphilitics. If we take that view we will approach the passing of the old hopeless epilepsy.

THE RESEMBLANCE OF THE SENSORY SYMPTOMS OF POST-DIPHTHERITIC ATAXIA TO THOSE SEEN IN THE CORD CHANGES OF SEVERE ANEMIA*

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PHILADELPHIA

Combined sclerosis or postero-lateral sclerosis of the spinal cord occurring in certain conditions, more especially pernicious or grave secondary anemia, has been recognized for many years; it was not, however, until 1913 that the characteristic loss of sensation was pointed out by Dejerine.¹ That eminent French neurologist showed that the affection of the posterior columns begins medially and spreads outward; as a result the fibers which are first involved are the long conduction fibers which ascend uncrossed in the columns of Goll and Burdach. This gives a loss of certain forms of deep sensibility, that is, of the sense of position and the sense of vibration; all other forms of sensation remain intact until the disease has produced widespread changes in the cord, changes which amount practically to a transverse myelitis. Patients with the disease almost invariably have subjective sensory disturbances in the form of paresthesias of the feet, legs and hands.

Diphtheria may involve the nervous system in a number of ways, thus the only sign that nervous structures have been attacked may be paralysis of the soft palate or of accommodation. The disease often produces serious disturbances of motion and coordination in the extremities. There is often a diffuse weakness or paralysis of the lower or upper extremities or of all four extremities. Another manner in which diphtheria may involve the nervous system is in the production of an acute ataxia which Dejerine² has described as a peripheral neurotabes.

From a pathologic point of view there is widespread involvement of the nervous system in diphtheria. Oppenheim states that "the diph-

* From the Department of Neurology, University of Pennsylvania.

* Read in the Section on Nervous and Mental Diseases of the American Medical Association at the Seventieth Annual Session, held at Atlantic City, N. J., June, 1919.

1. Dejerine: *Société de Biologie* 75:554, 1913.

2. Dejerine: *Arch. d. Physiol.*, 1887.

theritic poison acts upon the whole nervous system, producing the most marked symptoms now at this site, now at that, but most often in the peripheral nerves, and that it may also have a toxic effect upon certain areas without producing structural changes in them." He also states that the neuritic processes constitute the essential basis of diphtheritic paralysis. That this is true of post-diphtheritic ataxia I seriously doubt.

LITERATURE

A number of investigators have examined the nervous system in diphtheria. Some have studied material from persons who died after an acute illness, some from patients who died weeks or months after the attack of diphtheria and others have approached the subject from an experimental angle.

Preisz³ found a marked degeneration of the peripheral nerves. In the cord he found numerous hemorrhages, chiefly in the gray matter. The white substance was normal, except in one case which presented an advanced grade of degeneration in the posterior columns. Bikeles⁴ in one case of post-diphtheritic paralysis and ataxia found degeneration of the posterior column and to a lesser degree of the posterior roots. Arnheim's⁵ cases showed extensive degeneration of all nerves examined and capillary hemorrhages in the cord. Thomas⁶ found degeneration of the peripheral nerves, and in the cases where the cord was examined and in which a definite pathology was present, the posterior columns were always most seriously affected.

Henschen⁷ reported a case of acute disseminated sclerosis following diphtheria. His patient died three months after the attack. The microscopic study of the nervous system showed, in addition to a marked polyneuritis, a great number of disseminated areas of sclerosis in the cord, most marked in the posterior columns and associated with intense perivascular infiltration. Oppenheim⁸ and Redlich⁹ have had similar cases. In experimental work, Babes¹⁰ found general edema and softening in the cord most marked in the posterior and lateral columns. Martin¹¹ found the cord normal in his experiments.

There have been a number of clinical reports which show that

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3. Preisz: *Deutsch. Ztschr. f. Nervenhe.* 6:95, 1895.
 4. Bikeles: *Wien.* 2:26, 1894.
 5. Arnheim: *Arch. f. Kinderh.* 13:461, 1891.
 6. Thomas: *Reports of the Boston City Hospital*, 1898.
 7. Henschen: *Fortschritte der Medicin*, p. 529, 1896.
 8. Oppenheim: *Textbook of Nervous Diseases*, Chicago, Chicago Medical Book Co. 1: 321.
 9. Redlich: *Die Deutsche Klinik*, p. 561.
 10. Babes: *Virchows Arch. f. path. Anat.* 119:168, 1890.
 11. Martin: 1:26, 1892.

various parts of the central nervous system may be involved during an attack of diphtheria. Rolleston¹² investigated the plantar reflex in 870 cases of diphtheria. In 172 of these the extensor response was obtained at some period of the illness showing that the pyramidal tracts were involved either through a toxic action or by edema and capillary hemorrhage. The mortality was higher in the class of patients who developed the Babinski reflex and the incidence of paralysis was greater.

Taylor¹³ reported a case of Friedreich's ataxia which developed after a post-diphtheritic paralysis. In analyzing the symptomatology of that case it seems to me more likely that his patient really never recovered from the diphtheritic involvement of the central nervous system.

Tinel and Siredey¹⁴ reported the eighth case in literature of chronic bulbar paralysis following diphtheria. They explained the occurrence of the bulbar symptoms in their case by assuming that the toxins traveled along the nerves and ultimately produced sclerosis of certain nuclei of the cranial nerves.

AUTHOR'S OBSERVATIONS

The cases which I desire to record are of interest from the type of sensory loss which they show, the loss of sensation being exactly similar to that seen in the combined sclerosis of pernicious or severe secondary anemia. The subjective symptoms which were complained of in these cases were of the same character that is so common in anemia.

REPORT OF CASES

CASE 1.—A man, aged 22, had diphtheria two months before the onset of symptoms which were paresthesia in hands and feet, fatigue on exertion and difficulty in walking. Examination revealed a Romberg sign; loss of all deep reflexes; loss of the sense of position and vibration in feet and legs; marked impairment in recognizing the points of a compass; all other forms of sensation were intact.

History.—The patient, aged 22, a student in the University of Pennsylvania, came to the Students' Dispensary in the last week of February, 1917. During Christmas week of 1916, he had diphtheria, and was given three injections of antitoxin during the course of the illness which confined him to his room for ten days. He was apparently well until a few days before he came to the dispensary, two months after the attack of diphtheria. At that time he had numbness in the feet and hands. The numbness became gradually worse and

12. Rolleston: *Rev. Neurol. and Psych.*, p. 404, 1910.

13. Taylor: *Proc. Roy. M. & Chir. Soc., Sec. of Neurol.* 7: 1913-1914.

14. Tinel and Siredey: *Bull. et mém. Soc. méd. d. hôp. de Par.*, Nov. 22, 1906, p. 1126.

was associated with other paresthetic phenomena. The legs became weak and walking caused fatigue. He did not have either difficulty in reading or diplopia.

Examination.—The gait was ataxic and a Romberg sign was present. The biceps, triceps, patellar and Achilles reflexes were absent. Plantar stimulation produced flexion of the toes on both sides. The extensors of the toes and feet were slightly weak. Sensation was normal for light touch, pain (pin prick and the pain of deep pressure), heat, cold and deep pressure throughout the body. The sense of position was seriously disturbed in the toes and ankles; the vibratory sense was affected below the middle of the legs; there was impairment of the compass test over the feet and lower part of legs. There was slight ataxia and clumsiness in the upper extremity, without any disturbance of sensation or motion. The cranial nerves were normal. The nerve trunks were not tender on palpation. This man recovered entirely and when last heard of was with the American Expeditionary Force.

CASE 2.—*A soldier, aged 24, contracted diphtheria in France early in January, 1918; shortly after his throat cleared up, he had difficulty in reading; regurgitation of fluid from the nose; six weeks later paresthesias in the feet, soon involving the legs and hands; marked ataxia in all extremities; Romberg sign present; all deep reflexes lost; sense of position, vibratory sense and the compass test markedly disturbed in the feet, legs and hands; all other forms of sensation intact.*

History.—This patient was a soldier belonging to the 163d Infantry and stationed near Blois, France. In the first week of January, 1918, he had a severe attack of diphtheria for which he was treated at the camp infirmary for two weeks. Coincident with his discharge from the hospital he had dimness of vision and regurgitation of liquids through the nose. These conditions persisted three weeks and then gradually wore away. On February 20, he had for the first time paresthesias in the feet; the paresthesia became worse and soon involved the legs and hands. In a short time he had difficulty in walking and in using the hands for the finer movements, such as dressing and writing. He had no pain. He was sent to Blois on March 28.

Examination.—He was very ataxic in the finger to nose test and in walking and presented a marked Romberg sign. The biceps, triceps, patellar and Achilles reflexes were lost. Plantar stimulation produced flexion of the toes. There was no motor involvement with the exception of slight weakness in the flexors of the fingers and wrists. The cranial nerves and the pupillary reflexes were normal. Sensation was normal for light touch, pain (pin prick and deep pressure), heat, cold and deep pressure throughout the body. The sense of position was seriously impaired in the toes, ankles, fingers and wrists; the vibratory sense and the compass test were impaired over the feet, legs and in the fingers and hands. The nerve trunks were not tender on pressure. This patient was not followed up.

CASE 3.—*A woman, aged 37, had diphtheria in November, 1917; at Christmas, paresthesias in feet which in time involved legs and hands; difficulty in walking and in performing fine movements with hands; gait and station markedly ataxic; sense of position and vibration impaired in toes and feet; touch sense very slightly impaired over dorsum of feet; all other forms of sensation normal.*

History.—This patient was referred to Dr. Spiller by Dr. Guthrie of Sayre, Pa., and she was admitted to the University of Pennsylvania Hospital, Feb. 18, 1918. I am indebted to Dr. Spiller for the privilege of reporting this case and the following notes are taken from the hospital record. In November, 1917, she had diphtheria and was ill for two weeks. She was apparently well

from the first day out of bed until Christmas, when paresthesias were noted in the feet. In the course of three weeks the paresthetic sensations ascended as high as the knees. Two weeks before admission to the hospital, she began to have difficulty in walking and in using the hands for sewing and writing. She had never had pain in the legs or arms.

Examination.—The gait was very ataxic and a marked Romberg sign was present. There was no ataxia in the finger to nose test. The biceps, triceps, patellar and Achilles reflexes were absent. Plantar stimulation produced flexion of the toes on both sides. The arms and hands were moved about freely and no muscular weakness was present. The lower extremities were moved well but there was slight weakness in certain movements, namely, extension of the toes and feet and extending the legs on the thighs. Sensation was normal for pain, heat and cold throughout the body. Touch was slightly impaired over the outer side of the dorsum of both feet. The sense of position was lost in the toes of both feet, but was normal at the ankles. The sense of vibration was lost over the feet, and was diminished over the lower part of the legs. The nerve trunks were not tender on deep pressure. The cranial nerves, the reflexes of the pupils, the blood and the cerebrospinal fluid were normal. The urine showed a slight amount of sugar and on one occasion a positive test for acetone. This patient remained in the hospital only four days.

PRACTICAL CONSIDERATIONS

From an analysis of the sensory symptoms in these cases it will be noticed that all three cases had paresthesias in the hands and feet, and all showed a loss or marked impairment of the sense of position and of vibration, and that in two of the cases with absolute preservation of touch sense there was impairment of the ability to recognize the two points of a compass.

Cassirer¹⁵ has reported a case of post-diphtheritic polyneuritis with loss of osseous sense and the sense of position.

Where is the pathology of a process which can pick out and impair the conduction fibers for the sense of position and the osseous sense, and also render the recognition of the two points of a compass impossible or nearly so?

Head¹⁶ has given us the following information regarding the relation of the various forms of deep sensibility (with the exception of the sense of vibration) to one another in the peripheral nerves and in the spinal cord and also their relation to the compass test. The power of discriminating two points of a compass depends on the integrity of touch sense. If this is impaired it is clear that the compass test is of little use. In cord lesions studied by Head, especially cases of Brown-Séquard paralysis, the power of discriminating the two points of a compass was impaired or lost on the side of the motor weakness and was associated with loss or impairment of the sense of

15 Cassirer: *Die Deutsche Klinik*, p. 804.

16. Head: *Brain* 28:537, 1906.

position and passive movement. In the peripheral nervous system there is an intimate relation between sensibility to deep pressure and the pain produced by it, and the ability to recognize passive movement and position. All three are present or absent after lesions of the peripheral nerves and all three depend on the integrity of those fibers which run with the muscular nerves. In the spinal cord the fibers conducting the sense of position and passive movement are separated from those conducting deep pressure and the pain of deep pressure and ascend with the vibratory or osseous sense in the posterior columns.

LOCATION OF THE LESIONS

It seems reasonable to me to place the lesions which account for the sensory loss in the cases here described in the posterior columns of the cord and not in the peripheral nerves. So far as I know, in no other part of the nervous system are the fibers which conduct the osseous sense, the sense of position and the ability to recognize the points of a compass grouped together. In all of these cases the motor loss was insignificant and the ataxia was the predominating symptom. If disease of the peripheral nerves were responsible for such a loss of sensation one would expect to find in addition, a loss of deep pressure sense and a marked impairment of motor power.

Pathologic reports show that the posterior columns of the cord are usually most seriously damaged when the cord is involved during the attack of diphtheria. The cases of Bikeles, Henschen, Oppenheim and others show the same to be true in cases that recover from the acute illness only to succumb to the subsequent disease of the nervous system. In the consideration of the changes which occur in the central nervous system during an attack of diphtheria it is interesting to note that perhaps a number of cases of disseminated sclerosis may originate from this infectious disease.

The nature of the pathologic process which I have postulated as the cause of the sensory loss related in my cases is not necessarily destruction of tissue, but a toxic action on the posterior columns, and very likely in some instances due to edema and capillary hemorrhages which usually clear up.

THE DIAGNOSIS OF "WAR PSYCHOSES"

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Shortly after the beginning of the war it was rumored that a number of anomalous psychoses were being observed among the soldiers; that is, patients in whom all the symptomatology previously would have led the psychiatrist to give a gloomy prognosis, but who recovered spontaneously without apparent residual. This group was understood to contain only psychogenetically-determined psychoses, and did not include any cases which might be regarded as organic. Until the fall of 1918 no opportunity was offered us to see anything of this war result with the exception of a few sporadic cases.

Two of these, however, were calculated to increase one's interest in the type of clinical material to be seen. The first case was that of a psychosis in a sergeant who had led an unusually sheltered life, with no adequate notion of sex adaptation. He had had no opportunity frankly to face problems because his entire family life had been dominated by the Christian science activities of his mother. The psychotic episode followed pneumonia. After an initial period of delirium, the disorientation disappeared completely, leaving the patient with ideas that electricity was being placed in his bed; that he had been made into a monkey; that he had been contaminated with "filth," and that "purity first" would save the world. He complained of a sense of body alteration. Nocturnal enuresis persisted for some time, and was regarded by the patient as a fluid withdrawn from him by the influence of "some woman." All his utterances gave evidence of profound, instinctive disorientation in the face of clear, general orientation. Although the soldier had not seen overseas service, the case suggested what might be expected if men were suddenly transplanted from a sheltered, protected life, with its relatively simple environment, to the hurly-burly of active service, with all its difficulties of adaptation. After his recovery he gave an account of the horror with which he had viewed the facts and manifestations of sexual life as told in the barracks.

A second patient—an officer who had been discharged with a diagnosis of paranoia—when first seen presented an elaborate system of persecutory delusions with retrospective falsifications. The patient had a long history of difficulties and to each he had reacted with

gastric hypochondriasis. After a period of intense homesickness overseas, he was notified that he was to be sent to the front. The fear of active service, although existing some time, suddenly became acute. His inability to face the situation frankly resulted in his acute psychotic episode. He justified his fear by regarding himself as persecuted. Interestingly enough, the gastric complaints disappeared completely throughout the period of his psychosis, only to reappear promptly when recovery from his paranoid state had taken place. Nothing in the symptomatology gave any inkling as to the outcome except the especially trying circumstances under which it had arisen. This case again gave evidence that anomalous psychotic episodes were to be expected when difficulties were thrust overwhelmingly on an individual barely able to handle ordinary problems in an environment to which he was reasonably well adapted and accustomed.

Another observation was recorded while treating psychoneurotics at U. S. General Hospital No. 30 during the summer and fall of 1918, where opportunity was offered to study patients who had developed psychoneuroses in this country as well as those who had developed them overseas. The cases could be separated roughly into two groups: (a) those arising in this country which were akin to similar states seen in civil practice, and (b) those which were the result of the unusual stress of active warfare. The majority of the former group showed insidious onsets and exhibited fundamental adaptative difficulties (inferiority feelings, masturbation worries, undue attachment to parents, etc.) and their symptoms antedated the war. The second group, although including soldiers constitutionally inferior, appear to be men much better disciplined to living. Probably this contrasting condition was brought about by the psychiatric weeding-out process as practiced in the camps. The fact that the overseas cases arose largely in response to the one dominating desire—to get out of the war—explains, to our minds, the relative ease with which they were cured, i. e., removal to hospital solved their difficulties. On the other hand, it was manifestly impossible to accomplish the same results for the domestic group of patients. We had, then, in the minor psychoses a lead to what we might expect in the major.

It was, therefore, with great interest that we welcomed the opportunity to study cases at a final clearing station—as it were—in a general hospital which was devoted to the major psychoses. We began our study of the material at U. S. General Hospital No. 34, in December, 1918. Many of the patients had been in the hospital for several months awaiting discharge. They had, apparently, completely recovered and each succeeding week brought a large number of additional recoveries. The majority of these cases had come to us with a

diagnosis of dementia praecox, although some of the diagnoses had been modified at the more recent hospitals to "Psychosis, Undiagnosed." As critically as the incomplete case records would allow, it seemed profitable to review this group of cases to discover, if possible, by what signs a more adequate diagnosis could have been made, or what the disease picture showed which could have given the key to the correct prognosis in the cases of so-called "praecox." We were prepared to take issue with the former classification which insisted on crowding all cases of the great group of the psychogenetically-determined disorders into the one of the two great pigeon-holes, manic-depressive psychosis or dementia praecox. Our study of these cases has strengthened that revolt against any classification which fails to take into account the great group of psychoses that cannot be correctly labeled either manic-depressive insanity or dementia praecox. We have no quarrel with words, but we do feel that a large part of the inability of the psychiatrist to sum up the case in terms of observed facts has been due to the feeling that it must be fitted definitely into one group or the other. If we substitute affective and schizophrenic disorders and admit admixtures of the two states we have a system sufficiently labile to include all cases, and we are not bound to prognostications which the facts do not warrant.

In reviewing the cases we have come to the conclusion that the errors of diagnosis were to be explained along the following line: (a) The affective disorders were not recognized as such unless they existed in "chemically pure" form. (b) Schizophrenia, as a benign psychosis, was not admitted. (c) Acute fear reactions with confusion, hallucinosis, and sometimes disorientation were fairly uniformly regarded as praecox. (d) Little attempt was made to understand the cases as reactions in terms of personality, conflicts and wishes; that is, the "surface" content was not given due weight.

A. AFFECTIVE DISORDERS

In the group of the affective disorders was found, by far, the largest number of errors. Records will be presented to illustrate the factors which it is felt should have given the clue to diagnosis. Each case offered has received at least one diagnosis of "dementia praecox." No claim is made that the material presented is either original or startling, but justification for publication of these observations seems to lie in the fact that the course and outcome of these psychoses are not in accord with the impression which these cases originally inspired.

Among the affective reactions three groups seem to have escaped recognition. 1. Psychoses in which depressive ideas, i. e., self-recrimination, ideas of guilt and inadequacy feelings, dominated the picture. Two cases illustrate this type of disorder.

CASE 1.—J. A. W., man, aged 22, white, waiter, with nothing of interest in his personal or family history, when seen first at Camp Hospital, Nov. 23, 1918, was reported by a noncommissioned officer to have been restless and troublesome on the voyage to France. He had threatened suicide and refused to mingle with his fellows. His records state that he appeared depressed and moody, but there is no mention of the soldier's statement of affect. He said he had no confidence in his abilities and was unable to stand the strain of life. His mental processes appeared slow. On November 27, the patient was described as very quiet and talking only when spoken to. He appeared very nervous and spoke of worrying about his family and his own inability to make good in the army; he complained that he had no self-confidence. He answered questions slowly; there was no evidence of hallucinations or delusions at this time. On January 12, the patient stated that he had always been seclusive and not like other people; he felt inadequate and said he did not want to live because he was not smart enough; he was afraid that he was no good. When admitted to this hospital on Feb. 21, 1918, he gave a clear account of his illness. He thought that he was sent to the hospital because he could not get along, and because everything worried him. He attributed his trouble to a nervous breakdown. The patient was slow in speech and action, but gave no definite expression of depression. He has now completely recovered and shows no evidence of his old feelings of inadequacy, slowness of speech or action.

At no time should the psychosis have been regarded as ominous or schizophrenic. His slowness and lack of contact with people was apparently due to his inability to break through his depression while his talk and ideas were centered upon his own inadequacy. There is no evidence of projection or incongruity in the picture.

CASE 2.—P. M., man, aged 26, white, watchmaker, with one year's high school education, was excessively alcoholic and had a gonorrheal infection at 19 over which he worried a great deal. He was drafted June, 1918, and got along well while in camp. He was sent overseas in September, 1918. While there he drank rather heavily and on one or two occasions became boisterous, noisy, and impertinent to his company commander. He was admitted to the hospital Jan. 11, 1919, "in a more or less negativistic state, which persisted for eight days. He talked little or not at all and when he did, was much retarded. He had attacks of apparent unconsciousness but there were no convulsive movements. He was oriented for time, but was not certain as to place." He stated that he had some disease in his system and that he was afraid that if he had anything to do with women he might give them the same sort of disease. He was well behaved on the ward, but he had numerous ideas about diseases, which to his mind accounted for his slowing down. He thought syphilis was responsible for all his troubles, and appeared somewhat depressed. In this hospital, on March 21, 1919, the patient was slow in movement and in speech. He rubbed his hands together and paced up and down the ward. He looked worried and anxious, and feared that he may have infected someone with syphilis. He dreamed that he was at home and was no good. He said he could not stop worrying. The patient was correctly oriented and no memory defect could be demonstrated. He was very neat, kept his clothes clean and pressed and his hair was well combed. He responded to questions very slowly and appeared thoughtful and preoccupied. In April he was interviewed again and said he felt that he was not wanted here in

the hospital; that he had done wrong because he had not attended church when there were so many churches to go to. He said: "It's my own fault, it's all my own fault, I think that my trouble is caused by that disease which I have." When pressed for an expression of affect he replied, "I feel sad."

All the ideas expressed concerned the patient's own inadequacy and feelings of guilt. He accepted all feelings of guilt as the result of his own misconduct and did not attempt to make the world stand the brunt of his own feelings of incompetence. What was described as negativistic seems likely to have been a stupor-like state which is so frequently seen in profound depression.

2. Psychoses which showed all the mechanics of depression, that is, difficulty in thinking, slowness in stream of talk, inactivity, were mistaken for schizophrenic slumps with apathy.

CASE REPORT.—D. H., man, aged 24, white. After finishing college in 1917 had a "nervous breakdown" in which he had marked feelings of incompetency; became "morose," depressed and contemplated suicide. He felt he was unable to be of any consequence in life; that he could not concentrate on any subject, and had a subjective sense of confusion. Although interested in chemistry and agriculture, he could not force himself to seek a position in keeping with his training and attainments. He therefore took a job in a mill, earning \$14 a week. He had practically recovered when he was drafted into the service. Shortly after his induction into the army, his slowness returned. When seen in a hospital overseas on August 18, he was described as "showing marked lack of interest, with marked hesitation in answering questions; appeared dazed with clouding of consciousness and disorientation for place and time." (We are inclined to doubt the statement as to disorientation because the soldier recalls accurately all hospitals in which he has been.) He is said to have had much "disturbance of association" and retardation, as well as to have been actively hallucinated, although no description of hallucinations was given. A week later, at another hospital, he stated that he was no good to the army because he had not started right. He slept poorly and worried because he was "mixing everything up," and expressed a desire to die. His replies to questions were extremely slow; there was no spontaneous speech, and his slowness was described as "dulness and apathy." On admission to this hospital on Oct. 18, 1918, he is said to have appeared indifferent and apathetic. He was "pessimistic about his future, complained of lack of confidence, and could not see how he could be of any use in the world." In February his speech and actions were still slow. In the early part of March he was more cheerful, and worked as clerk in the canteen, but still gave expression to his inferiority feelings. When interviewed again in the latter part of March he had a subjective feeling of difficulty in thinking, looked worried, was slow in talk, and described his mood as "somber." He showed a morning-evening variation in his difficulty of thinking and slowness of speech. At that time he gave a history of his early youth. He has always been sensitive and has worried a great deal about masturbation. He had nocturnal enuresis until 14 years of age, but this disappeared after an operation for varicocele. He felt, as a boy, that his masturbation had permanently injured him, and that it prevented him from developing a strong will or a fixed view in life. He realized, as he grew older, the part that sexual ruminations have played in his inability to reach out to a mature standard, but showed nothing

suggesting projection or abnormal submersion of his auto-erotic difficulties. At the present time the patient has practically recovered. He is cheerful and is in sole charge of the canteen from time to time.

While the patient's type of personality suggests fertile soil for a schizophrenic development there had apparently always been sufficient affect to swing him along. His slowness and difficulty of thinking appear to have been mistaken for apathy. His ideas at all times were consistent with his own feelings of inadequacy. The failure to see the case as an affective disorder was due to the fact that the affect was not on the surface and the mechanics were not given sufficient weight. Many case records show this same failing. Patients were repeatedly described as apathetic and disinterested because their reactions were slow; few records contain mention of affect and practically none give the patient's own words concerning his mood and feeling. In the next group is seen the tremendous importance of mood deviations as a leavening factor in the course of psychoses in which projection phenomena are prominent.

3. In affective disorders with schizophrenic features too little attention was given to the emotional setting in which the psychotic development took place.

CASE 1.—P. C., man, aged 27, a white laborer who had reached the fifth grade in school had a negative family and personal history as far as is known. He enlisted May 6, 1917, but for some reason was not sent overseas. In June, 1918, while in camp he developed measles, making an uneventful recovery. A short time after the infection he began to tire easily, his speech became slow and difficult, he could not sleep, was restless and became apprehensive and suspicious for no apparent reason. He was reprimanded by a noncommissioned officer for slowness and was told that the former would hypnotize him in order to put life and action into him. In October he was granted a pass to go home. On his arrival he felt peculiar; his eyes seemed green, and he felt as if they were being drawn together. He attributed this to the fact that someone had hypnotized him and was exerting an influence over him. He thought that this person was someone in his company. Therefore he returned before his pass had expired to discover who it might be. He complained at this time of a heavy and sluggish feeling about his heart. Soon after his return he began to feel that he could hypnotize others; that the men in his company were talking about his actions; that they disliked him and were going to try to poison him. In addition, he felt that he was being talked about and that many remarks were being made behind his back.

He was admitted to this hospital Dec. 18, 1918, giving at that time a clear account of his illness. He appeared depressed and said that he felt "sad." It was with difficulty that he could be made to smile and he spoke in a slow, hesitating manner. He complained of difficulty in thinking. He was well oriented and his appearance was neat and tidy. On February 15, he was discharged as completely recovered. His depression and slowness had entirely disappeared, and he realized that his ideas had been wholly imaginary. At no time previous to admission to this hospital had any notation been made concerning his mood.

We have, then, a reaction which would have appeared as extremely ominous, i. e., with ideas of reference, influence and a sense of body alteration, had there been no frank affect; but the definite statement of sadness, with his slowness and difficulty in thinking made the outlook relatively benign from the beginning.

CASE 2.—P. A., man, aged 23, white, with meager education and an insane mother, is said to have had "fear attacks with ideas of impending death" at the age of 5 or 6. He also is said to have had "spells"; there is nothing in his later history, however, to suggest epilepsy. He has shown a roving disposition, being unable to settle down to any one job for any length of time. In fact, he has had so many different kinds of work, and each for so short a period that he is unable to name his occupation. He was admitted to a hospital overseas on Aug. 21, 1918. There he was described as follows: "He appears dull and not very much interested in environment, talks slowly and ideation is slow." Said "someone is trying to take my thoughts away, and that the company commander was saying things about him. He also felt that he could tell what other peoples' thoughts were. Said he had been doped. He is rather sad and has loss of emotional tone." Two weeks later at another hospital he said, "They have tried to give me poison to do away with me because they thought I was a German." He admitted hearing voices but there was no statement as to the content of his hallucinations. He was sorry that he had talked so much. On admission to this hospital, Oct. 7, 1918, he appeared depressed; said that he was "taken for a spy and a German"; he felt that his company thought that he was trying to do things detrimental to them; that the night watch could tell about him as he slept. At one time he imagined that poison had been put into his food; also that his thoughts were being read. He stated that he had been depressed, but felt better when interviewed. On discharge, in March, he spoke of his psychosis as having been "imagination and junk." His depression had entirely disappeared and he denied any ideas of influence, reference or persecution. His old roving disposition reasserting itself, he went away without leave on a number of occasions, but while in the hospital did excellent work in the shoeshop.

Although the affect was in the background, it gave sufficient evidence that his ideas of influence, reference and persecution, together with his apparent disinterestedness, were to be regarded as less ominous signs than one would ordinarily be inclined to believe.

CASE 3.—X. Z. Certain facts which might serve to identify the patient are omitted for obvious reasons. The patient is a young woman who was reared in an austere, religious atmosphere, in which dancing, card playing and theaters were strictly forbidden. She describes herself as sensitive and self-depreciatory; for example, she could not understand what her husband had seen in her to have married her. She admits that she always had a tendency to day-dreaming and found it easier to build air castles than to obtain her satisfaction from reality. She was married a year and a half ago, and one month after marriage noticed a vaginal discharge which was diagnosed as gonorrheal, but no smear was made to confirm the diagnosis. A sense of modesty prevented her from taking her husband into her confidence. As it had been her greatest ambition to bear children, she worried tremendously for she feared she might be sterile. When her husband went overseas she threw herself into war activity with great fervor and enthusiasm. Toward the end of March she began

to feel tired and attempted to bolster her strength by drinking large quantities of coffee. Her friends noticed that she seemed very happy, and she describes her mood as one of elation. No amount of work seemed too great for her. A short time previous to onset she had read a story in one of the current magazines about automatic writing. (During the period preceding her elation she had some notions that her physical fatigue might prevent her from being in fit condition to bear children on her husband's return. Her husband's mother had been opposed to her marriage.) She decided to make a trial with automatic handwriting and discovered that through this medium she could commune with God and her husband's father, to whom she had been greatly attached and whom she nursed during his last illness. She was told that if she was humble in spirit and pure in heart she would have many children and bring joy to her husband. She soon found that it was no longer necessary to use handwriting as a medium because she could communicate directly with God, Jesus Christ and the Holy Ghost. She was found, on March 31, lying naked on the floor; she appeared unconscious, although she stated after her recovery that she knew definitely what was going on the entire time. Her reason for this action was that she had been told that if she washed herself and appeared humble in the sight of God she would have many children. "When she was examined the next day she was 'loquacious and euphoric.' She said she had been dead for several hours during which time she was in the presence of Jesus Christ; that she had purified her heart and been born again. She refused to divulge the nature of her interview with God, and insisted that the examiner was not pure enough to receive the information. She became angry at the examiner when he asked her if she had made any efforts to prevent conception and stated that she could read his brain and knew that he meant to offend her. Her attitude at that time suggested exaltation. She laughed during the examination and gave the impression of elation."

On admission to this hospital she showed practically no signs of any psychosis. She gave a clear and cogent account of the episode and after a few interviews, in which an attempt was made to point out the relation of the content of the psychosis to her longing and ungratified wishes, she accepted the point of view, was able to understand what each of her ideas had stood for, and abandoned all notions of the reality of her spiritual communications.

This fantastic flight into spiritualistic and occult phenomena with ideas of mind-reading, exhibitionism and eroticism is the material out of which schizophrenic development is ideally woven. However, all of the content of the psychosis when viewed in the light of the elation and exaltation takes on a benign coloring.

B. SCHIZOPHRENIC DISORDERS

The next group, that is, benign schizophrenia, comprises a much smaller number of cases, but does include a sufficient number to warrant their presentation. We place in this grouping patients whose symptomatology is in no obvious manner different from those cases which form the classic group of dementia praecox. We see, in the schizophrenic development, a mechanism which Bleuler has designated as "autistic thinking," i. e., a withdrawal from reality, into a world of fantasy, with projection of that part of the personality, which is not

tolerable, onto the external world. The extent to which this withdrawal takes place, with the necessary internal readjustments which appear objectively as bizarre activity and thought, determines the profundity and seriousness of the disorder. The factors which make for good are the patient's ability to be aroused to normal emotional responses, to be able to understand the psychosis in terms of conflicts and precipitating causes. The precipitating causes of the withdrawal are potent factors in estimating the hope of return to the real world of fact and recovery. In the war cases the prime motive for this flight from reality could, in the large majority of cases, be removed, and it is our impression that it was principally in those cases in which the conflict was closely associated with the instinct of self-preservation and the inferiority complexes brought out by the war, that startling recoveries were noted. Again, we do not claim that there is anything new about the recognition of this group. The war, however, did give us "stress and strain" in condensed and powerful form, and therefore performed for us, as it were, a large number of "experiments."

CASE 1.—D. M., negro, aged 38, steward and cook who drank alcohol in moderation, gave a negative family history with gonorrhea at the age of 33. After his graduation from high school he became a steward in a private club, and went into the catering business for himself seven years later. He failed after three years' trial and gave a history of a nervous breakdown at that time. He was "nervous and suffered from indigestion." We have no record of any more serious psychotic disturbance at the time of his failure. He spent a few months at Hot Springs; made an uneventful recovery and returned home, securing employment as a waiter.

At the age of 32 he married. The marriage was unhappy and his wife finally secured a divorce, after numerous attempts at reconciliation had been made. The patient maintains that his wife was unable to satisfy him sexually. In November, 1917, he enlisted, and was sent overseas in April. While still in this country he was "nervous, dissatisfied, and had stomach trouble." He had trouble with the men of his company because of his attitude of superiority and his higher education. In May he wrote his company commander complaining that the other men in his company were jealous of him and "had gotten on his nerves." He asked to be sent to penitentiary as life was miserable for him, and stated that his only desire was to get to the front to earn a commission. (He was serving as a mail clerk in a labor battalion.)

When admitted to a hospital he thought that the adjutant disliked him and that a sergeant had mistreated him, making threats against him. He maintained that a roentgen-ray picture was taken of his head to show that he was insane. He regarded his transfer from hospital to hospital as an attempt to get him out of the way. He thought he overheard officers and other patients talking about the commission which had been given him. Although he admitted that no one had ever told him directly that he had been given a commission, he insisted that many times he had heard people say, "That is the man with a commission." He thought that there was some plan to get up a political campaign against the governor of his state, and he was told to prepare speeches against it. He then developed the idea that machines had been put

around his head to obtain his speeches. When examined at this hospital he insisted that he had been given a commission in the Medical Corps and asked for permission to go to Washington to discover why the commission had not arrived. He felt that certain army officers and high officials were against him. On one occasion he heard the voice of a woman calling him from the second floor of the Administration Building; later he thought that a system of mind-reading had been adopted to prevent his leaving the hospital. He spoke of having been given three commissions, medical, infantry and quartermaster; he said that everyone could tell what he was thinking by mind-reading and that the other patients on the ward "tapped out" his thoughts. His explanation for receiving a commission in the Medical Corps lay in the fact that "medicine comes natural to me." On one occasion he offered to fight an officer if he would remove his bars because he had been asked what kind of a mason he was, and felt that there was some hidden significance to the question. On the street he heard people remark, "There is monkey-face; they are making a monkey out of him." At all times he was correctly oriented, his memory was good, and he gave no expression of affect that showed any departure from the normal. He was always neat and orderly, and took excellent care of his clothing. Military manner and bearing were well maintained, and he worked willingly at whatever work he was asked to do. When allowed to leave the post on pass, he returned promptly and never got into any trouble. On several occasions he was persuaded that his ideas were imaginary. Finally, after a furlough in which he renewed his contact with his old friends and interests, he abandoned these ideas completely and came to recognize them as a response to his old feelings of inadequacy and discontent, and as the fulfillment of a wish.

At first his delusions, passivity feelings, ideas of reference and tendency to misinterpretations made one uneasy as to the outcome of the psychosis, but the fact that he always worked readily, was interested in the activities of the post, was careful about his personal appearance, and that there was little tendency to rumination and day-dreaming with inactivity, made us hopeful that there was sufficient material on which he could rebuild.

CASE 2.—I. F., private, white, aged 23, clerk, single, had nothing of interest in his family or past history. About the first of December he began to feel sick. In the morning when he arose he felt dull and dizzy; he complained of seeing things double and had pains all over his body; he slept poorly and could talk only with difficulty. He was given aspirin for several days, but did not improve. Finally he was sent to a hospital, but his physical examination showed no abnormalities, and at no time was there any sign of an acute infection. When seen in the hospital he showed marked indifference to his surroundings, lay for hours in one position, and held his body rigidly. He resisted any attempts to change his body position, but when finally moved maintained any position in which he was placed. He was well oriented, giving adequate response to all questions which were asked of him. He wet the bed, had to be spoon-fed and drooled saliva constantly. After three weeks he improved, but gave a meager account of his illness. He said he thought that he had been paralyzed. At no time was there any evidence of delusional or hallucinatory experience. On admission to this hospital March 7, he had recovered completely. He recalled the facts of his illness, but could give no explanation for his psychotic episode.

One would have suspected *a priori* that many such "acute catatonias" would be seen, but up to date very few have been observed in this hospital, and this case is the only one that has made a recovery. The absence of projection phenomena and the acute onset might have suggested the benign course of the episode. At first the case gave the impression of an obscure, infectious process, but the drooling of saliva and the incontinence in the absence of any confusion or delirium would seem to exclude this possibility.

CASE 3.—L. K., private, aged 30, machinist, moderately alcoholic, single, with a negative family and personal history, was born in Russia, and came to the United States in 1909. He worked regularly as a machinist, earning as much as \$3.25 a day. He was drafted Oct. 7, 1917, going overseas in June, 1918, although he saw no active service at the front. On Sept. 16, 1918, he was admitted to the hospital complaining of pain in the chest which he thought was the result of poison put into his coffee. He thought that the sergeant in his company was against him, also that the men in his company had been influenced against him. For this reason they undertook to poison him. He thought that gas and electricity had been put into his bed; he heard that his money was to be taken away from him. On awakening one morning, he told the ward attendants that there was blood around his umbilicus; that his blood had been spoiled; that syphilis had been given him, and that his semen had been drained away. He imagined that his picture was being taken wherever he went. After a few days his delusions appeared to quiet down, but one night he awoke and accused the nurse of trying to poison him, and said that electricity was put into his bed to make him jump. There was no frank fear except that exhibited by refusal to go to bed because of electricity or to eat because of poison.

When admitted to this hospital, Dec. 6, 1918, he was disinterested, and on questioning gave the same delusional system as obtained overseas. No expressions of affect could be elicited and the soldier's expression or appearance gave no evidence of fear. Feb. 18, 1919, he had made a complete recovery. The mental status at this time showed no abnormality except that he claimed amnesia for the period while in France. He worked at various jobs about the post and said that he felt better than when he entered the service.

It is a striking fact that a large number of cases seen in this hospital have occurred in soldiers who are either foreign born or who cannot speak and understand English adequately. At present sixty of the 240 patients in this hospital are either foreign born or have serious language difficulties. A possible explanation for the large number of such psychoses may lie in the fact that foreigners are usually the object of jokes and bantering. Unless associated with others of the same race, they were apt to have little social contact and in many instances were ignorant of the causes of the war, and what they were fighting for. It is probably true that the general material from which immigrants are drawn is below par; at any rate, the observation is striking.

These three cases illustrate, in relatively pure form, what has been seen in fragments in a much larger group. The impure form contains

delirious elements, initial periods of confusion and bewilderment with various admixtures of delusional and hallucinatory projections. The content of these psychoses concerned itself with fundamental instinctive disability, and gave evidence of profound conflicts. It cannot be too strongly emphasized how important it is to recognize the benign nature of many of these disorders; all that there was of confusion and delirium should have been noted as favorable. Much of the hallucinatory experience was on the surface and concerned itself with visions of home and family, the voices of friends and relatives.

C. ACUTE CONFUSIONAL, HALLUCINATORY DISORDERS
WITH FEAR

These impure forms graduated into a group which seemed sufficiently well defined to be set off by themselves, i. e., the cases of acute confusional, hallucinatory episodes with marked fear. It is true that we have little understanding of this group which has been called toxic-infectious in the presence of definite, known etiology and "amentias" in its absence. While the content of many cases of this group was very much on the surface, a number showed reactions which suggested that the very roots of the personality had been stirred. Many cases showed considerable affect so that, had there been no records of initial disorientation and confusion, one would have classified the cases as affective. Stupors were quite common in this group.

CASE 1.—S. C., corporal, aged 29, white, mill hand, was alcoholic until 1916, but has not used alcohol since. His mother is said to have been nervous. He had only a meager education. In 1914 the patient had a "nervous breakdown" lasting three months, at which time he was "very nervous"; had sensations of "flashes" passing through his body and involving his spine; was restless and unable to work. He made a complete recovery from this attack. In June, 1916, he enlisted and was sent to the border. He became nervous occasionally but did not have to go to a hospital on this account. He was sent overseas, and after a short time his organization moved to the front. In July, 1918, it is reported that he was knocked over by a shell, became unconscious, and was sent to a hospital. When seen at the casualty clearing station, he was confused, could give no account of himself, and had a "wild and anxious expression." His confusion disappeared after a few days, but he complained of weakness and his talk was rambling and "irrational." He was transferred to a hospital in England and there showed considerable emotional instability and some perplexity. He thought he was going to be killed, that electric batteries had been placed in the ward to kill him, and that his grave was being dug outside. He was recommended for discharge on a diagnosis of dementia praecox. When admitted to this hospital, Oct. 7, 1918, the soldier was entirely well. He recalled his feeling of intense fear, but claimed that he had no memory for the initial period of confusion or rambling talk.

CASE 2.—R. G., private, aged 24, oil chemist, indulged in alcohol to intemperance at times, but gave no history of excess overseas. Prior to enlistment

he had done excellent work as a commercial chemist. There was no history of any previous mental trouble. After arriving overseas he was sent to the front, and there was a history of his having been sent to a hospital several times for hypochondriasis. On Nov. 20, 1918, after having been lost for about a week, he was discovered wandering aimlessly about, and when taken to the hospital gave a history of having had a headache and pain in the legs for a week previous. He had been unable to sleep. Three days later he became fearful, easily startled, thought he was to be injured and that soldiers were pursuing him in order to shoot him. He said his food tasted queer. He was completely disoriented and was described as apathetic, slow, retarded and irritable. His "memory was poor, and he was untidy." On Dec. 7, 1918, the patient rushed out of the ward, saying, "they are after me." He secured an axe and struck a prisoner over the head, fracturing the skull, and accused the prisoner of having killed his father. He continued to refuse food, saying, "you have me now." While being evacuated to another hospital he tried to escape in order to avoid his persecutors. His disorientation continued, and he ran up and down the ward banging his head against the wall. He persisted in his delusions about his food and the attempt to kill him. He was untidy and incontinent, and gave the impression of great fear. His sleep was disturbed by terrifying battle dreams. On admission to this hospital, Jan. 10, 1919, he appeared confused and depressed. He complained that he lacked ambition; was unable to concentrate, and thought that his memory was poor. At this time, however, he was correctly oriented, and showed no evidence of fear. His talk and response to questions was very slow, and he was not readily accessible. The depression and slowness, however, gradually disappeared and he was discharged in March as completely recovered. His mental status at this time was normal. He showed correct orientation, no affect, and complete insight into his delusions and psychosis. He was alert, bright, showed no memory defect, and was ready to take up his job as a chemist.

A question, which for army purposes is of great importance, arose in this case, and gave us warning that one should accept facts of the history, taken during the psychosis, with caution. He gave a definite history that two years previous he had had a similar mental upset. His family, who were most anxious to cooperate, denied this, and were unable to give any facts that might have been construed as a psychosis. On his recovery the soldier also denied any previous breakdown. Obviously "line of duty" questions are seriously involved, and one should in justice to the soldier, accept only such history as seems beyond peradventure.

CASE 3.—M. P., private, white, garage worker, whose father was moderately alcoholic, also drank in moderation. He was a "delicate child" and had "fits" from the age of 5 to 12. He reached the sixth grade in school. He was sent to France in December, 1917, only to develop "stomach trouble" as soon as he arrived overseas. He was on sick call constantly and in July, 1918, developed tremors, growing more and more "nervous." In August he became confused, disoriented, losing himself on several occasions. His statements while in the hospital were quite inconsistent; he spoke vaguely of having been deafened by the noise of motors and appeared frightened and anxious. He was lachrymose, inattentive, seclusive and unresponsive to questions. His condition was much improved after three weeks and he asked to be sent back to his outfit.

This request was made at an evacuation hospital where it was generally understood that soldiers were not returned to the line. When seen at this hospital, Oct. 7, 1918, he had completely recovered. He gave a clear account of his illness and verified by definite statements the fact that he was fearful.

CASE 4.—G. A., private, aged 31, mill hand, white, born in Italy, was moderately alcoholic and, until his present illness, was never in a hospital. He was drafted in April, 1918, and was sent overseas the next month. Throughout his army career he worked in the kitchen, and did not participate in any engagements. He gave a history of having been gassed in October, 1918, but the medical officer who observed him first made no note to this effect. When first seen he was confused, wandered aimlessly about, was unkempt and dirty. He appeared anxious and frightened. A few days later he gave a history of having had bad news from home and of having worried a great deal on this account. The voices of his mother and sister kept calling to him to come home. On occasions he became frightened and attempted to escape from what he thought was a jail. His talk was rambling and fragmentary. He wept frequently and developed an irregular tic of head and facial muscles. Later he gave a history of constant fear that something terrible was going to happen to him. On several occasions he was disoriented but the disorientation does not seem to have been constant. When admitted to this hospital, Jan. 21, 1919, he was correctly oriented, alert, gave a clear account of his illness, showed no affect, and there was no evidence of confusion. He was discharged ten days later as completely recovered.

It is interesting to note that "gassing" has become as respectable a cause of mental disease as "shell-shock." Almost every recovered case until the signing of the armistice gave gassing as one of the causes of the illness.

To summarize the foregoing groups it may be said:

1. The affective disorders were unrecognized in the cases in which (a) ideas of guilt, self-recrimination and inadequacy statements dominated the picture, rather than frankly expressed depression; (b) the mechanics of depressions, i. e., difficulty in thinking, slowness in the stream of talk and inactivity were the prominent features; (c) schizophrenic elements were present. Too little weight was given to the emotional setting of this group.

2. Benign schizophrenia was not correctly prognosticated because: (a) The acute onset was not taken into account; (b) the patient's capacity for activity and interest was ignored; (c) there was no realization of the fact that the existing cause of conflict could be more readily removed than is usually the case; (d) the patient's ability to understand the psychosis and its causes, and his ability to make necessary readjustments was underestimated.

3. In the acute confusional hallucinatory psychoses with fear, too little weight was given to the elements of confusion, delirium and affect.

The foregoing cases were selected from a large number, all of which were diagnosed as dementia praecox.

Two other groups of cases seem sufficiently interesting to deserve comment. First: a surprisingly large number of acute paranoid developments have been seen. Some have appeared with affective background, and others without. The occurrence of paranoid trends has been very frequent throughout all the war psychoses as observed here. One is led to accept the statement of Bleuler that paranoia is not the unusual disorder that institutional material would lead us to believe. Some of our cases which were originally on an affective basis have settled down to a delusional system without any affect, and this group lends some weight to the theory that paranoia is primarily a mood disorder. Other cases lend no support to this view and seem to have shown a pure "intellectual" disturbance from the beginning. The following two cases are chosen from a group of ten or twelve.

CASE 1.—C. T., coal and ice dealer, aged 27, had reached the seventh grade. He was moderately alcoholic, but not to excess at any time and had been nervous a year and a half before entering the army. About three years ago he had a breakdown which lasted for three months. He was afraid to leave his house at this time, was very "nervous," apprehensive and feared that he was going to die. He had cardiac palpitation, general body tremor, and showed great restlessness.

He was drafted in September, 1917, and was sent overseas in April, 1918. In August he became suspicious, thinking that he was being watched. He felt that the relatives of a married woman whom he had known illicitly in the states were in his company and were planning to do him harm. He began to watch the men about the kitchen. His own statement at that time was as follows: "They acted suspicious, and seemed to be laying for me. They were always watching me—watching my movements. I had the cooks with me and they (the conspirators) were apparently waiting for them (the cooks) to go away. They stayed all day, just watching and waiting for me. Then they returned to the company. That night I told my captain and he advised me to get a rest. The next day I was sent to the hospital, and the same thing started there. At night two men sleeping on each side of me flashed lights in my face and turned their wrist watches at me to show where I was lying. This kept up for four or five days. I could see them talking in conversation. They always appeared suspicious to me because they were all in one bunch. They sent me away to another hospital because I was afraid that they were plotting against me. There another gang started. They laid to get me. There is one fellow here who came with me, and I am suspicious of him. He started the trouble and there is somebody on my trail all of the time. It is because I had trouble back home over a married woman. Her husband started it back in the states. I was all right until two weeks ago. Then they found me here and started the gang after me. I am not getting a square deal, they follow me wherever I go and they are going to kill me. They bother me day and night so that I can't sleep because of them. They are always whispering about me. There is a conspiracy on to get me. The Italians are in it." The patient was said to have been apprehensive and suspicious, and to have shown considerable fear. He looked about anxiously. His records, however,

give no statements of the patient, himself, concerning fear. He could not be persuaded to abandon his delusional system and insisted on being evacuated to the states to avoid his pursuers.

On admission to this hospital, Oct. 7, 1918, the soldier showed no signs of fear, suspicion or apprehension. His behavior seemed entirely normal and he said that, while he did not fear his persecutors while in the army, he had an idea that they would try to persecute him in civil life. He could not be convinced that his suspicions and fears were imaginary. His attitude at this time was one of placidity. He talked freely about his delusions, but showed no fear of any sort. He fell into the hands of one of the other patients, and after a few days he asked to be allowed to appear before the disability board again. He seemed to be entirely converted by his friends, and stated that his ideas were imaginary, having no foundation in fact. While awaiting discharge he was questioned repeatedly and each time stuck to his position that the whole affair was psychotic. Persistent attempts to lead him to accept his delusional system again, were unavailing.

It is impossible to be sure whether the statements about fear refer to a dominating mood or whether fear was the result of the ideas of persecution. His delusional system at this hospital, however, stood free from any mood disorder.

CASE 2.—M. C., aged 28, butcher, Italian, nonalcoholic, was born in Italy, and came to America in 1908, after four years' schooling in Italy. He was drafted in October, 1917, and went overseas in August, 1918. While working as a laborer he had some trouble with the men with whom he was associated. He thought that some of the men in his company were going to knife him or use a razor on him, he could not sleep because of the fear of being killed. He appeared very fearful and apprehensive and declared that he was no good; that the other soldiers were talking about him, and that he was going to be killed as a result of this. On October 21 he was described as talkative and excitable. He told the same tale of persecutions and admitted hearing voices telling him he was to be killed because he was no good to the army. There was no evidence of depression. When admitted to this hospital, Oct. 25, 1918, he showed no abnormality in his behavior or mental status. He denied any ideas of persecution, influence or reference. No expression of affect could be elicited. He showed little insight, however, but was willing to admit that his ideas were imaginary.

The second group is heterogeneous in the sense that it includes all types of reactions: schizophrenic, affective and confusional, but possesses this one common feature—the initial period shows symptoms of the so-called psychoneuroses. In our work we have followed Dana's and Meyer's notion of speaking of major and minor psychoses. The cases in which hysterical, psychasthenic, neurasthenic and anxiety symptoms occur in pure form shade insensibly into the fullblown psychotic developments. The army workers have attempted to separate the two groups—psychoses and psychoneuroses—probably because of the desire to prevent "the stigma of insanity" from being attached to patients whose adaptive breaks did not lead to total incapacity. When dealing with the facts of cases as we can understand

them—that is, of reactions of psychobiologically integrated organisms—we believe that no essential difference in mechanism, or even in the severity of reactions, can be made out on any large scale. Hysterical delirium may appear as *praecox*; anxiety neuroses may assume the ranks of agitated tension states with complete major psychotic pictures; psychasthenic symptoms may be mixed with profound depression, and neurasthenoid symptoms may progress to deteriorating schizophrenias. Such a phrase as "the insane psychoneurotic" shows the futility of attempting a classification of patients into these two groups which aim only at reaction tendencies. At this hospital, supposedly devoted to the insane, we have seen every conceivable variety of mixture of symptoms. At U. S. Army General Hospital No. 30, which was supposedly devoted to the psychoneuroses, a somewhat similar situation prevails. This, according to our notion, was not due to the carelessness of previous examiners, but followed as a corollary of this false division. We present two cases as illustrations. The first one seemed clearly hysterical at the onset, but apparently assumed a more automatic character, and readjustment took place at a deeper level. The second case was also hysterical and superficial at onset, but matured into a major psychosis with features that were distinctly ominous.

CASE 1.—R. G. B., aged 30, painter, moderately alcoholic, who had reached the eighth grade, entered the service April 3, 1918, and was sent overseas in June. Early in August he was sent to the hospital, because of complaint of sharp pains in the cardiac region, palpitation and rheumatic pains in the left leg. He had occasional fainting spells with sensations of blackness before the eyes. A few weeks later he showed no improvement in his somatic complaints, and in addition began to show a lack of interest in his surroundings. He sat by himself and could not be aroused to any activity. He insisted that his heart had stopped beating and that his lungs had collapsed so that he could no longer breathe. He thought that his food did him no good. No expression of affect was elicited. When evacuated to this country he was described as "seclusive and showing a marked interest defect." When first seen at this hospital in October his apathy and disinterestedness continued, and the original diagnosis of dementia *praecox* was confirmed. At the end of November he was re-examined, and examination at this time showed him to be normal. He was alert, cheerful, interested and without complaint. He said he felt as well as ever. He stated that he was nervous before entering the army, and admitted that when he was taken sick he had just heard that his company was going to the front.

CASE 2.—E. A. B., aged 22, white, moderately alcoholic, had a brother who had been discharged from the army on account of major hysteria. He had had periods of undue worry and has always been regarded as having a nervous temperament. These periods of slight worry and depression have usually been followed by slight exhilaration, but have not been sharply enough differentiated to suggest a cyclothymic process. He graduated as a dentist, but was unable to pass the state board examination. He was forced to marry his wife because

she was illicitly pregnant. The girl, however, was acceptable to him and to his family. While in the service he worked as a dentist, although he was not commissioned. Early in November he received letters from his wife saying that she was not getting on well with his family with whom she was living. She also wrote that she was pregnant, and thought she would die. This caused him some worry, and he felt that he ought to be back in the states. He arrived overseas in September, 1918. In the latter part of November, 1918, he had three attacks characterized by fainting with convulsive movement, but without loss of consciousness, sphincter control, or injury. He himself characterizes these attacks now as "hysterical." He feels "sure of this because they resemble attacks my brother had." He was classified as an epileptic and recommended for transfer to the United States. These attacks appear to have been very near malingering, or at least to be definitely in relation to a desire to return home. He knew that his brother had been discharged from the army on account of hysteria. While in the hospital his worry and depression increased. He began to think that people looked at his feet, and by this they meant that he was a sex pervert. He also thinks they called him a sex pervert. While en route from Tours to Savonay the idea developed that he had heart disease and paresis. He felt that he never would get well.

On examination here, March 21, 1919, it was found that he was alert and answered questions promptly and relevantly. He avoided discussion of his seizures, but when confronted with the notion that they caused his evacuation to the states and served a purpose he did not deny it, but smiled acquiescence. He wept when his "sexual perversion" was discussed. He gained control readily and laughed at his instability. His family insisted that he had always been as unstable as the foregoing indicates.

Although the emotional instability and lack of adaptability indicate that we were dealing with a constitutional inferior, the transition from the surface psychoneurotic episode to an affective psychosis with ideas of reference is evident and suggested an ominous coloring.

CONCLUSIONS

Our study of some of the "war psychoses" revealed that:

1. Affective disorders were frequently mistaken for dementia praecox.
2. Psychoses showing typical schizophrenic development had recognizable benign features.
3. Acute confusional hallucinatory psychoses with fear were incorrectly diagnosed as dementia praecox.
4. Acute paranoia was a relatively common psychosis.
5. The distinction between psychosis and psychoneurosis is untenable.

We wish to express our thanks to Lieut.-Col. William H. Smith, M. C., U. S. Army, for permission to study these cases, and to Dr. Adolf Meyer of the Johns Hopkins University for his advice and kindly criticism.

Abstracts from Current Literature

THE SYMPTOMS OF ACUTE CEREBELLAR INJURIES DUE TO GUNSHOT INJURIES. GORDON HOLMES, M.D., *Brain* 40:461-536 (Part IV, Dec.) 1917-1918.

As a catalog and dictionary of the signs and symptoms of acute cerebellar disease, this article can be judged to be as complete and satisfactory as is possible at the present time. While there do not appear any marked contributions to our knowledge of cerebellar function, yet it serves a useful purpose in its minute and painstaking analysis of the disturbances which may follow injury to the cerebellum. There must perforce still remain much work for the "hewers of wood and drawers of water" in the future before we are able to identify the real inwardness and essence of the organ in question. In clearing the ground for further advance and clarifying our thoughts on this most difficult of neurologic problems, the work of Gordon Holmes should be sure to sow seed which shall be reaped at some future time.

The article, following the custom of the more pretentious contributions to *Brain*, is divided into an introduction and chapters. The first chapter is devoted to a most minute analysis of the disturbances as studied in a number of unilateral cerebellar wounds received in the past war, and comprises over one half of the space covered by the entire article, the remainder of the article being concerned with wounds of the vermis, bilateral injuries, wounds of the cerebellar nuclei, the nature of the symptoms produced, the chapter being directed toward an understanding of the underlying principles, and finally a chapter on cerebellar localization.

There are a number of illustrations and graphs showing tracings of movements as influenced by cerebellar damage.

In the introduction the author emphasizes the difficulty experienced in reconciling his clinical studies with the results of experimental investigation on account of the impossibility of correctly and accurately estimating neighborhood effects and the infrequency of clear-cut pictures. His studies were based on over forty individuals, twenty-one of whom were studied over relatively long periods of time. The greater number of cases presented unilateral damage.

Chapter 1, on unilateral lesions of the cerebellum, is divided under eleven subheadings:

(a) Disturbances in Muscle Tone: The motor side receives the main effect of cerebellar damage, and tone in particular suffers. The muscles are soft and flabby on palpation; they can be displaced, compressed or stretched without pain or discomfort to the patient. The normal resistance to passive movement is lost, and movement is limited only by bony architecture and ligamentous check action. Although the movements are allowed abnormal range and resemble those seen with flail joints, there is no ligamentous relaxation. This loss of tone is uniform throughout the limb or limbs, the result being as great distally as proximally. It appears early, from five to ten days after the receipt of the injury, and may persist for several weeks. Decrease in the tonelessness is uniform, there being no anisosthenia which was sponsored by André-Thomas.

(b) *Disturbances of Voluntary Movement:* There is present a definite asthenia, slowness in initiating contraction and relaxation as shown by graphic records, and a much greater expenditure of energy to accomplish a definite end as compared with the normal side. Dynamometric readings show a decided loss of power in even the simplest acts where the factor of awkwardness would play the smallest part. A more prompt fatigability appears, and manifests itself in irregularity and jerkiness in the application of the force; this may be attributed to the voluntary efforts to overcome the results of fatigue. Static strength that is the maintenance voluntarily of position seems unaffected. There is a decided reluctance to movement of the affected limb which is present even in the stage of excitement in anesthesia. Electrical stimulation of the muscles shows no loss in the promptness in muscular response, and there is no lengthening of the latent period after the application of the current.

1. Delay in initiating muscular contraction.
2. Delay in attaining the exertion of the full force.
3. Delay in commencing relaxation.
4. Delay in effecting relaxation.

All muscle groups are affected uniformly; there is no limitation of the range of movement, no rigidity and no contractures.

The ataxia present may be studied to the best advantage early before compensation by other motor mechanisms takes place. Vision plays no part in cerebellar ataxia. Movement is less prompt; the member sways and deviates from the proper line, the movement is less accurate, is performed with undue force and the ataxia is increased by haste. These results seem to depend for their existence on the disturbed action of the nonmotofacient musculature, the fixating muscles at the joints.

An analysis of the ataxia shows:

1. *Decomposition of movement.* Improper coordination of the various components of a movement.
2. *Asynergia.* Failure of coordination between the agonists, the antagonists and fixating muscles.
3. *Dysmetria.* Failure of the check element which again is lack of coordination between synergists.
4. *Tremor.* Usually at the end of a movement and resembles intention tremor of multiple sclerosis, again irregular coordination of synergistic muscle groups.
5. *Deviation from the line of movement.* Failure of the nonmotofacient components.

The more complex the movement, the greater will be these phenomena.

The rebound phenomena receives a special place of its own, although it is merely another example of the asynergia, the loss of the check element.

Adiadochokinesis is manifested by an inability to perform alternate movements in rapid succession, the rate, range and regularity of sequence being lost. Instead of the orderly alternation of contractions, there appears an awkwardness and irregularity due to the disturbed action of antagonistic and synergistic muscle groups and the addition of unnecessary adventitious elements. The tonelessness of the muscles and the slowness of contraction and relaxation contribute their shares. Contrary to the normal, there is a greater ease in performing movements with one hand at a time instead of with both hands synchronously. This is equally true of coarse and fine movements.

(c) *Static Tremor*: The swaying of the head and body present in cases of cerebellar injury is due to irregular and discontinuous muscle action. The posture of the limbs once attained is usually well maintained and, in fact, it may be better sustained than on the normal side, which recalls the state known as "cerebellar catalepsy." The fine, vibratory tremor normally present is absent. With fatigue, a coarse tremor appears which is due to the effects of the increased fatigability of the muscles and the efforts toward voluntarily resuming the ordered attitude.

(d) *Vertigo*: This is present usually in early cases, the rotation being from the affected toward the normal side. The patient lies on the affected side with the head rotated toward the unaffected side and the ipsilateral shoulder advanced, which may be interpreted as a latent tendency toward rotation.

(e) *Spontaneous Deviation of the Limbs and Bárány's Pointing Tests*: If the eyes are closed, the affected arm will almost uniformly swing slowly outward, and past pointing will always follow the same trend.

(f) *Attitude*: This feature, so striking in experimental animals, is much less marked in man. The head is retracted toward the affected side, but rotated toward the normal side; the body is concave toward the affected side, and the ipsilateral shoulder is advanced. The ipsilateral limb influenced by gravity will be abducted, rotated outward at the shoulder, and pronated at the elbow.

(g) *Standing and Gait*: The patient will sway toward the affected side, with the head and trunk inclined in the same direction and the spine concave toward the injured side, the weight being mainly carried on the contralateral leg. The ipsilateral shoulder is advanced. The patient, if pushed, will fall more readily when he is pushed toward the affected side. There is no Romberg. If the attention is distracted, standing becomes more difficult. In the gait there is difficulty in maintaining the equilibrium, stumbling and falling toward the affected side. In walking, the patient constantly tends to deviate toward the side of the injury. The affected leg evidences a tendency suddenly to fail the patient, and will give way most unexpectedly.

(h) *Disturbances in Ocular Movement*: These are present only early after the injury, and consist in a deviation of the eyes toward a "rest-point," which lies away from the affected side. Movements toward the affected side are slow and performed with considerable effort. A skew deviation was observed in five cases. A nystagmus of fixation is present, more pronounced in looking toward the affected side, the eyes tending to drift away from the point of fixation and toward the "rest-point," only to be brought back by the sharp volitional component. The rate is about twenty-three to thirty jerks per 10 seconds. There is usually no nystagmus on vertical gaze or during convergence. It is more rapid, finer and less regular when the eyes are directed toward the unaffected side. The nystagmus increases the farther the gaze is directed away from the rest-point.

1. Nystagmus of fixation, slow phase toward the rest-point.
2. Increased by looking toward the affected side.
3. Gradually decreases.

4. Differs from labyrinthine nystagmus, which is present at most for only a few days; the slow phase is always toward the affected side, and by the fact that cerebellar nystagmus is increased by lenses which cut out fixation.

(i) **Disturbance in Speech:** Speech is slow, drawling and monotonous, sometimes staccato, scanning or explosive. Phonation is more affected than is articulation.

(j) **Reflexes:** These are diminished, being more feeble, less brisk and elicited with greater difficulty. Summation is often necessary before the reflex can be obtained. The latent period of 0.043 second is the same in the normal and the affected limb. The fall of the limb is much retarded, owing to the retarded relaxation of the quadriceps extensor. In the normal leg the ratio stands as follows: Rise: fall: 1: 1.2 or 2.2, whereas in the affected limb, rise: fall: 1: 0.85. Owing to the absence of tone and the check elements, a series of oscillations result before the leg comes to rest, the pendular knee-jerk of André-Thomas. As a result of the absence of the check element of the hamstrings, if the patient is lying in bed with the plantar surface of the foot on the bed, there is absent the normal resumption of position after tapping the patellar tendon, and the leg can be completely extended by a series of taps on the tendon. The superficial reflexes are unaffected.

(k) **Sensation:** This is absolutely unaffected.

Chapter 2 deals with symptoms due to lesions of the vermis. The only definite observation on this style of injury was a greater difficulty in phonation and articulation, more pronounced tremor and a greater difficulty in movements of the head and trunk.

Chapter 3 is devoted to symptoms of bilateral lesions of the cerebellum. Both sides show the results of injury, as outlined above under the head of unilateral symptoms. Speech is very much affected, being slow, drawling and often almost unintelligible. The muscles of the head and neck are very hypotonic, and the gait is seriously affected.

Chapter 4 discusses symptoms produced by lesions of the cerebellar nuclei. As it is impossible for these nuclei to be injured without a concomitant injury of the cortex, clear-cut pictures are impossible and we can only draw deductions between superficial and deep injuries. In the latter the symptoms are more intense, more regular and disappear less rapidly. The symptomatology differs only in degree.

Chapter 5, on the nature of the symptoms produced by cerebellar lesions, recapitulates and reviews the signs and symptoms enumerated above with an interpretation. The constancy, regularity and persistence of these symptoms incline one to believe that they are destruction symptoms. Vertigo is probably the only irritation symptom present. The early areflexia is probably due to the early extreme hypotonia.

1. Atonia. A constant, important and striking result of cerebellar injury.

2. Asthenia.

3. Retardation of movement, delay in initiation of contraction and relaxation. The cerebellum is probably a motor reinforcing pathway, and exerts its effect on the motor cell of the final common pathway.

4. Discontinuity and irregularity of muscular contraction. Sudden relaxation, astasia, incomplete fusion of elementary muscle twitches.

5. Ataxia: (a) asynergia; (b) decomposition of movement; (c) deviation from the line of movement.

6. Dysmetria, attributable to pathologic slowness in commencing contraction and effecting relaxation. Defective check element.

7. Speech defects—asynergia.

8. Eye movements. Asthenia prevents the maintenance of fixation with consequent deviation toward the rest-point.

9. Attitude.

Cerebellar function is probably active on the afferent side, integrating proprioceptive impulses and maintaining through the final common pathway the readiness of muscles to contract and relax.

Chapter 6 discusses functional localization in the cerebellum. Careful observations at the time of operation, deductions as to the probable course of the injurious agent, roentgenograms and necropsy experience in the author's cases shed no light on functional localization in the cerebellar cortex. Injury, no matter how slight, affected the entire side. All portions of the cerebellum except the antero-inferior surface were found affected, yet no limitation to any body segment was ever observed. The single exception was that injury to the vermis seemed to affect profoundly the musculature of the head, neck and trunk. He could find no support for Bárány's cerebellar localization.

RILEY, New York.

EXTRAMENINGEAL MENINGOCOCCUS INFECTIONS. W. W. HERRICK,
Arch. Int. Med. **23**:409 (April) 1919.

This paper is based on the study of 315 cases of meningococcus infection which Herrick observed as chief of the medical service at Camp Jackson. In 40 per cent. of the cases the diagnosis was made before meningitis developed. Five per cent. did not develop meningitis. Most of the cases in Herrick's series showed signs of a general infection hours, days or weeks before the meninges were involved; this initial stage of sepsis was proved by blood cultures and necropsies. The stage of sepsis averages forty-eight hours before there is a local manifestation of the disease which is usually in the meninges, but often elsewhere. Herrick quotes six cases in which meningitis was absent or played a subordinate rôle, the predominating clinical pictures being sepsis, poly-arthritis, pleurisy or empyema of the accessory nasal sinuses.

Herrick makes a plea for a change in name of the disease commonly called cerebrospinal meningitis. He recommends that meningococcus infection should be used to designate the sepsis which the intracellular diplococcus may produce and that meningococcus meningitis should be the term for those cases in which the meninges are involved in the local process.

This paper is extremely important and interesting. If Herrick is correct in his premises the most vital fact to be learned is the additional avenue of treatment, i. e., the intravenous injection of serum.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

Three Hundred and Seventy-Second Regular Meeting, April 1, 1919

WALTER TIMME, M.D., *President*

FACTS AND FANCIES IN PSYCHOANALYTIC TREATMENT. Presented by DR. A. A. BRILL.

Dr. Brill stated that there are many reasons at this time why he chose this subject, chief among them being the fact that though psychoanalysis had become a well-known and popular subject in many walks of life, many persons, even those in the highest standing in neurology and psychiatry who were sympathetic to psychoanalysis, often showed a marked ignorance of its basic principles. It should not be forgotten that this form of treatment did not lend itself to the amelioration of acute forms of neurosis; it was, for instance, as impotent in removing so-called *shell shock* as it would be in removing hernia. Psychoanalytic treatment had its limitations, and the cases selected for this form of therapy should not only be persons of normal intelligence and of good character, but they must be over the acute attacks.

The chronic psychoneurotics of normal mental make-up furnished the best cases and some of the most profound hysterias and compulsion neuroses had been cured after every other form of therapy had been tried in vain. Most of these people showed some sexual difficulty. Looking over the records of the cases sent to him during the first years of his practice, Dr. Brill found that over 60 per cent. were referred to him just because the patient spoke of sex or his physician expected it. An enormous amount of nonsense had been said and written about sex psychoanalysis. To understand the strange manifestations frequently found in man, for example, abnormal fancy formation, or the actual perversions, etc., one had to be aware that the sex impulse consisted of many components and partial impulses which were congenital and developed with the individual. To the cultured person sex stood for much more than the sexual act. Conceived in this broad sense, one had no difficulty in comprehending the existence of an infantile sexuality and in realizing that no neurosis, even in a child, was possible in a normal sexual life.

A great many cases sent to psychoanalysts could easily have been cured without this if the family physician knew something about the cause and effect of psychosexual disturbances. A great many physicians, especially those dealing with nervous patients, also needed to cultivate with them a better *rapprochement* similar to that cultivated by psychoanalysts and designated by them as transference, a mechanism of approach concerning which there was considerable misunderstanding. This mechanism of transference was put into operation by the patient continually applying to the physician hostile or tender emotions which had no foundation in the actual relation, but were derived from the patient's unconscious fancies. The transference had to be managed with a great deal of tact and skill, for there was a tendency in neurotics with a floating libido to be ever ready to fix it on some one, identifying that person with the

good father who spoiled them or with the lost lover, etc., which was absolutely impossible in the relationship existing between patient and physician. The same mechanism was constantly found in normal life and on it was based attraction or repulsion.

Since many cases that were sent for analysis were not psychic, it was essential that one practicing psychoanalysis should have a thorough knowledge of the mechanism of hysteria and the other psychoneuroses, as well as a good knowledge of neurology and psychiatry. Only those possessing these qualifications should prepare themselves for psychoanalytic work. It should not be forgotten, also, that a pathologic condition could exist in a neurotic individual, and no analysis in the world would remove symptoms due to an organic disease. Thorough diagnosis of each case was of enormous importance before resorting to therapy, and therefore it was obvious that this was no subject to be played with by laymen. Psychoanalysis was in the strictest sense a part of mental medicine and deserved the sympathetic encouragement of the profession everywhere.

A PSYCHOLOGIC STUDY OF SOME ALCOHOLICS. Presented by DR.
L. PIERCE CLARK.

The speaker presented this paper at this time because of the seriousness of the modern psychologic aspect of the problem, and through a realization of the fact that the influence of alcohol on modern life was not to be disposed of merely by bringing about prohibition. In excessive and habitual indulgences in alcohol there lay a multitude of causes and results for study and analysis. At one time alcohol might serve to bring about harmonious relations in a social group otherwise difficult; at another it might aid to bring about a state of pleasant *rapprochement*; while at still another it might make easy for free egress deeper and illy adjusted unconscious motives. Alcohol was dangerous only to those who used it for illegitimate means and ends. In many instances these alcoholic individuals had failed to complete certain emotional cycles of earlier development wherein alcohol prevented proper and satisfactory repression or socially acceptable sublimations. Extreme alcoholic repression called for its precise study and analysis and no less insistently than did alcoholic indulgence; it was possible that the ardency of the prohibitionist was a compensatory public inhibition for more intimate personal liberties denied or repressed. It was often popularly held that a man totally abstinent in one field might be licentiate in another. It might be well that the alcoholic suppressed his libido and only freed it in drunkenness, while a neurotic expressed himself sexually. As eminently practicable as legal prohibition might seem at this time, it must not be forgotten that a signal increase in all sorts of neuroses and psychoses would come about as a result of such measures. Neither individual nor social alcoholism would be cured by prohibition; these could be effected only by careful analysis which by uncovering the fault would open the way to applying a remedy.

All the conscious motives given for alcoholic indulgence were but specious casuistry, or at least inadequate rationalizations. To arrive at any true analysis of the defect, not only the conscious reasonings had to be considered, but an investigation conducted by all methods possible of the unconscious strivings met or perverted by alcoholic indulgence. The use of alcohol was proportioned to one's idea of its value, but the habitual alcoholic was especially tormented with and guided by strong and overweighted feelings and representations, the real roots of which lay in the unconscious. In many cases of dipsomania the homo-

sexual component was quite transparent, shown in the wish to "treat" other men. The fear and restlessness which introduced the so-called dipsomaniac attacks were usually rooted on conflicts and repressions of the homosexual. Starting from the fact of bisexuality of human beings, both of organic and psychosexual attributes, surely the homosexual component must show itself in some way; if it could not be done openly and nakedly, then masks and symbols must be used. It showed itself in different degrees of sublimation. It was not merely chance that men so much enjoyed being among themselves and drinking together. Why did the alcoholic deliriant always see certain animals which were well known as sex symbols in general, and, especially seen by men as showing homosexual designs? Many alcoholics also had a "reverse Oedipus" complex which showed in deliria.

Unconscious homosexuality was only one factor in the alcoholic psyche; atavistic reminiscences played a large rôle. Atavism occurred in both healthy and abnormal states, but especially in the alcoholic one came close to atavistic remains, and chronic intoxication awakened and cleared the way for the ancient relics. Atavism gained new life in those psychically sick, and the drunken man exhibited unmistakable evidence of the sadistic-masochistic complex. The sadistic component explained numerous delinquencies and crimes that accompanied alcoholism. Alcohol numbed the higher functions. All drinkers did not become criminals, but alcohol certainly permitted hidden criminal desires to work out. The sexual component alone did not explain the behavior of alcoholics; the whole psychic content had to be considered. The unconscious had different levels or depths, and in different degrees of intoxication various levels of unconscious strivings and conflicts were released.

This conception of alcoholism afforded a scientific insight, made obvious the innate fault of the instinctive life, the fixation in the evolution of the emotional life, and showed the pattern plan of what sort of training out and social readjustment was necessary to heal such individuals. Anything less in the way of a comprehensive treatment was doomed to an early failure. In the definite periodic drinker the character usually showed less of the epileptic constitution *per se* and more of the instability make-up of the constitutional inferior. Altogether the study showed the truth of the contention that the line of treatment must always rest on the individual and social analysis of the particular subject under consideration, and that here, as in other profound neuroses, psychoanalysis might be undertaken. In the majority of cases one might hope for an arrest of the habit if proper precautions and lessened social demands were made on these special types of inferiors.

DISCUSSION

DR. HORACE W. FRINK opened the discussion of these two papers on various aspects of psychoanalysis, taking up first the question of alcoholism in which, though his psychanalytic experience had not been very extensive, it had been very unhappy from the therapeutic standpoint. He found that whenever he reached an important and difficult place in the analysis the patient got drunk and repeated this almost indefinitely, and he had never yet been able to finish an analysis of an alcoholic. On the whole, the things Dr. Clark pointed out were more or less confirmed by the speaker's own experience; in some of his cases the homosexual element was prominent, in others apparently almost negligible. There were different types of alcoholics; three in particular. One was the true periodic drinker, a condition related closely to the fugue and to cases of alternating personality. Such types were sometimes observed among clergy-

men and other people of a pious turn of mind in their ordinary state, but who showed in periodic sprees an exactly opposite set of characteristics. Then there were cases of mild psychoneurosis in which drinking began as a drug is taken to allay the fears or depressions, but at length became part of the neurosis. Again, there was a type that was largely chemical, the man who drank through the external psychologic influences that were present and got up a state which called for new doses of alcohol to overcome it. Some of those cases had been successfully handled with hypnotism, which was the only variety of treatment that had been in any way successful in the speaker's experience.

Regarding Dr. Brill's paper, Dr. Frink recalled ten years ago at a meeting of the County Medical Society that Dr. Fisher read a paper on psychotherapy. The president of the society had prefaced the discussion by stating that psychotherapy having been in existence for some time it was time for final opinion and judgment to be formulated. The various methods were discussed, particularly the psychoanalytic, and much invective and sarcasm were let loose against it. For some time it was all one's life was worth to mention psychanalysis at a medical society meeting, to say nothing of reading a paper on the subject. But as time went on, through the efforts of a few sincere men who had to endure much in the way of insults and antagonism, psychanalysis had at length become more or less respected. The battle for its right to exist had apparently been won. On the other hand, in a way, the battle had just begun, for the greatest danger was now presenting itself, in the form of the undpopularity of psychanalysis and its practice by the untrained, to which Dr. Brill had alluded. A serious factor, and one largely responsible for this popularity, was the fact that it was being practiced by those who bore no medical degree and possessed no medical education. There were many objections of the most serious character against psychanalysis being practiced by nonmedical people. In the first place, a nonmedical person, even supposing he had a good understanding of psychanalysis per se, could not safely be trusted to practice. A physician was something more than a person who had listened to lectures on medicine and studied medical textbooks. In securing his medical education, in his hospital training, in his association with his professors and fellow practitioners, in the hours spent in studying his cases, something of the ancient ideals and traditions became embodied with the personality of the physician which was not to be found in the layman, an attitude toward patients and toward society in general. The patient of the layman, however well trained in psychanalysis, missed that something which was very important.

There were still more practical objections of the greatest significance. To do good work in psychanalysis was enormously difficult; it took years of the hardest kind of application and study to achieve anything approaching decent success. One could not learn to analyze such an enormously difficult thing as the human mind without every advantage and the layman had no such advantage. It was to be regretted that there were certain physicians who not only had worked with these nonmedical people, but had sanctioned and endorsed their working alone and had referred patients to them. Such physicians surely could not believe they were doing the right thing by these patients, nor fail to realize that these patients not only were not helped but seriously harmed. A physician who sent patients to people of this sort was prostituting medicine for some sort of pecuniary profit. There was still a further consideration and one that should give pause to those who regarded this situation lightly. The lay psychanalyst was not bound by medical ethics as was the physician, and medical ethics constituted a protection to the patient which he should be given.

A final objection: the practice of medicine by the nonmedical was against the law, and it was time that this situation was investigated from that end, for it was getting to be a menace and if more of it went on, the good work that had been done and the repute that had been won for psychoanalysis by the serious workers would be in jeopardy. Psychoanalysis was not Christian Science and could not be regarded in the attitude one could accord Christian Science. It was not a religion; it was a difficult and serious medical procedure, involving medical examination, medical diagnosis and psychotherapy by a trained physician.

DR. C. P. OBERNDORF endorsed what Dr. Frink had brought to the attention of the society as to the necessity for medical training in undertaking psychoanalytic work. The question of selecting cases for analysis was sometimes a most difficult one, as there were many borderline cases. In some cases attempts at analysis would not only do the patient no good but might be harmful. This was especially true in certain cases of depression of the manic-depressive type where, during the active stage, any interference seemed only to accentuate the patient's sufferings without in any way relieving them.

So far as the speaker's experience with alcoholics went, he believed all that Dr. Clark had said was true psychologically. Alcohol furnished a convenient means of relaxation, but it was of most value in acting as inhibitory agent over the conscious mental censorship. Because of this, thoughts and actions ordinarily kept guarded were allowed to find egress. Possibly it was because of this being more or less realized and as such was contrary to one's best interests, that so many excuses had been unearthed for alcoholic indulgence. The relaxation which alcohol afforded was a very desirable one for the community. Prohibition would be a mistake, for though a substitute would probably be discovered, thus far none less harmful had been found.

It was very interesting to note the different types of individuals who found overindulgence in relaxing agents necessary, as they could be observed in the wards of Bellevue Hospital. In the alcoholic ward one would find the northern races, a frank type of people; in the drug ward was found the clandestine individual of the southern races. Many of both types would explain that it was from an unconscious urge they took the drug or drink. If alcohol was taken from these unbalanced individuals the tendency to adjust the emotional defect through drugs or other relief might be great. There was also great likelihood of an increase in neuroses and psychoses. The prohibitionist was putting the cart before the horse when he stated that alcohol was the cause of so many mental disorders; rather was the mental disorder the cause for taking alcohol.

Regarding Dr. Brill's paper, the crux of all psychoanalytic treatment lay in the handling of transference and resistance, and that was difficult to adjust at times. Transference had most of its roots deeply planted; unless unconscious resistances were eradicated analysis could do little good, and unless the analyzer accepted them as the really vital part of his problem, he did not accept his task at all.

DR. B. ONUF said that he had been so interested in Freud's work that he had been among the first to bring it to the attention of this society and to the profession at large. There were a few points that the discussion this evening had stimulated him to take up. In the first place, if there was any one entitled to give an opinion of psychoanalysis, it was Dr. Brill, who was what the Germans called the *alt vater* of psychoanalysis in the United States, and he was qualified in more than one way to practice it, first and foremost being his experience in

psychiatry. Dr. Frink's warning as to the peculiar fitness for psychoanalytic work was absolutely in order. Nevertheless, all knowledge of it should not be kept within the medical profession, but it should be spread at large because it was always true that when medical methods were known to the people they acquired a sympathetic understanding of them which promoted a larger application, not possible when kept within a small circle. There was no doubt that if an understanding of psychoanalytic theories was spread among the educated, great help would be gained in applying psychoanalysis to the proper purpose. However, there was one thing about psychoanalysts; it took a man's whole time, and it required special qualifications, an enormous amount of patience, a fine tact and other requisites that made for success, to say nothing of a good medical education. Dr. Brill stated that there was no harm in using psychoanalysis in dementia praecox even if there should be no success; this was true in the case of an experienced psychoanalyst, but there was danger in the hands of those whose experience had not been sufficient. He had known a case of dementia praecox in which one attempt at suicide after another followed certain revelations in the analysis. Dr. Clark had stated that most of his cases were constitutionally inferior, but many of the dipsomania cases were really cases of manic depression and the alcoholism was a part of the disease.

DR. HYMAN CLIMENKO asked what type of cases should be sent for special study and treatment to the psychoanalyst. Psychoanalysis had come to be an important part of medicine, but did it cure symptoms or did it cure hysteria? Which were those that could be cured by the shortest route and at the least expense to the patient?

DR. RUSSELL G. MACROBERT said that Dr. Frink's very modest characterization of his results with alcoholics tempted the question, because he knew how careful and painstaking his work was, if he did not consider that the psychogenic symptoms were not only attempted adjustments to real difficulties, but very many times and especially in more serious cases like pathologic drunkenness and early dementia praecox were the most satisfactory adjustments to his real difficulties that the patient could be contented with or was likely to find through psychoanalytic treatment alone.

DR. MICHAEL OSNATO asked if the impression he had received this evening was true, namely, that psychoanalysis had retired from the field of psychiatry altogether. Dr. Brill did not think it effective in the treatment of dementia praecox, which totaled more than 40 per cent. of all mental cases in institutions, and Dr. Oberndorf admitted that in manic-depressive psychoses psychoanalysis had little therapeutic effect, and these cases totaled from 10 to 15 per cent. of all admissions. They must of necessity express great reserve regarding psychoanalysis in general paresis, which totaled 15 per cent. of admissions; they spoke discouragingly of psychoanalysis in paranoia, which totaled 2 per cent. They must admit the same nonsuccess in senile and presenile psychoses. Dr. Frink and Dr. Clark had spoken discouragingly of the psychoanalytic treatment of the alcoholic psychosis, which totaled about 15 per cent. of all admissions to hospitals for the insane. In fact, they definitely admitted their lack of success in handling these cases. Now was one right in believing that psychoanalysis had entirely retired from the field of psychiatry?

DR. JOSHUA H. LEINER asked under what category the dipsomaniacs who were epileptics and who had a real organic base, according to Oppenheim and Aschaffenburg, came in their relationship to psychoanalysis.

DR. BRILL said that although his own experience with psychotherapy in alcoholism had been more successful than that related by Dr. Frink, he had nevertheless found the chronic alcoholic, as a rule, to be more or less deficient mentally, and whenever he succeeded in curing the alcohol craving something worse took its place which changed the individual from a menace to himself to a menace to society. Concerning the treatment by psychoanalysis of dementia praecox, Professor Freud had always maintained that these cases should not be analyzed because the praecox mechanisms in themselves were their abnormal adjustment, and in analyzing them that adjustment was destroyed. As a matter of fact, the adjusted praecox must eventually be sent to a sanatorium, and the disturbed praecox must also remain in the sanatorium. Incipient cases of praecox, however, could sometimes be benefited by psychoanalysis; and last but not least, a great deal of insight was gained by studying these cases, which was a great factor in prophylaxis. Dr. Climenko had asked what cases should be sent to the psychoanalyst. The answer was that only chronic cases that had resisted all treatment should be sent for analysis, although it would be better for the cases to be analyzed as early as possible. Dr. Osnato's query whether the psychoanalyst had retired from the field of psychiatry was surprising; psychoanalysts were psychiatrists first, and psychoanalysis was only a branch of psychiatry. The answer to Dr. Leiner's question was that no attempt was made to treat by psychoanalysis anything organic, but even organic cases like paresis and senile dementia showed freudian mechanisms in their delusions.

Dr. Clark was very glad to hear Dr. Brill's answer to the question as to whether psychoanalysts had retired from mental medicine. One of the great advantages to psychiatry had been the psychoanalytic method of approach, particularly in the benign psychoses and even in acute cases of manic depression. Properly applied in appropriate cases, it prevented the recurrence attacks. It also gave an excellent understanding of the mechanism of dementia praecox and a better insight into what to do for these cases through occupational and social readjustment, helping them even if they continued to follow a dementia praecox career throughout life. As regards its application to cases of alcoholism, psychoanalysis did not show up brilliantly, but in it lay a method of approach as to the mechanism controlling the condition, and the only way one could do any permanent good was by way of influencing the unconscious motives in those types of cases that were clever and resourceful people who did not lack in intellectual power. It gave an insight into the mechanism and how to help them make their adjustments.

As regards the relation of dipsomania to epilepsy, as quoted by Dr. Leiner, this conception was a great mistake; the dipsomaniac was more closely allied to constitutional inferiority rather than epilepsy per se.

NEW YORK ACADEMY OF MEDICINE
Section on Neurology and Psychiatry

Meeting held April 8, 1919

HYMAN CLIMENKO, M.D., *Secretary, presided*

CASE OF DYSTONIA MUSCULORUM DEFORMANS. Presented by Dr. S. PHILIP GOODHART.

Dr. Goodhart presented this case, as rather typical of a rare condition, for confirmatory opinion. It was the first case of its kind in his private practice, although he had seen a few at the Montefiore Hospital. The patient was

a girl, aged 24, whose family and personal history showed no taint. The onset of the present condition was atypically sudden and its progress rapid. The young woman had none of the stigmata of neurosis, and she was not emotional. Under unusual emotional stimulus, as for example the present demonstration, the spasmodic hyperextension of the muscles became more pronounced and beyond her control. The will had but little influence in the control of the movements, which did not persist when the patient lay quietly recumbent.

Dr. Goodhart said that such cases had heretofore been regarded as functional, but it was now quite a general opinion that they belonged to a definite group whose pathology might reasonably be placed in the corpus striatum. This important ganglion, deeply seated within the hemisphere, was the center of tone; and its component structures, with their connections with red nucleus and cerebellum, represented the extrapyramidal influence of the several phases of muscle tone. The case presented was distinctly a disorder of that function peculiar to physiologic muscle activity comprehended by the term *tone*. Dystonia musculorum deformans, a condition originally described as such by Oppenheim, was essentially one in which part of the musculature was in a hypertonic, another in a hypotonic, and another in a normal state of tone. This variation of tone became manifest on movement only. The same set of muscles might be involved, at one time hypertonic and again hypotonic. Characteristic was the hypertonicity of the leg muscles and those of the trunk and pelvis, so that in walking the trunk appeared to revolve about the pelvis.

DISCUSSION

DR. WILLIAM M. LESZYNSKY considered that the only objection to the diagnosis would lie in the suddenness of onset of the condition. Dystonia musculorum deformans was a gradually progressive disease.

THE SURGERY OF CRANIOCEREBRAL WAR WOUNDS AND ITS RESULTS. Presented by DR. HAROLD NEUHOF.

Dr. NeuhoF related facts drawn from his experience during the spring and summer of 1917 when he had the opportunity of treating patients with head wounds at U. S. Base Hospital No. 2 (New York Presbyterian Hospital Unit) attached to the British forces. These patients, the majority of whom had been operated on in the front area, fell into two classes: one in which there had been primary closure, and the other in which there had been drainage. The large majority of the first class did well, but sometimes secondary operations were necessary. When the dura had been lacerated by the missile or bone fragments a hernia cerebri was apt to develop, and such hernias often meant early acute brain infection with later development of brain abscess. Once a hernia cerebri appeared, hope for the ultimate outcome was not good. Concerning the second class of cases with wounds drained or incompletely closed, the results were on the whole unsatisfactory. The best ultimate outlook in serious head wounds existed after complete operations performed at hospitals in the advanced zone; operations short of complete were followed frequently by serious and often by fatal complications; and delayed primary operative procedures performed at the base generally had disastrous sequelae.

Accordingly, Dr. NeuhoF concluded, when sent with a neurologic team to British Casualty Clearing Station No. 17 in Flanders in the fall of 1917, that complete operations should be done, whenever feasible, on all head wounds. It was realized that efforts at complete operation might result in an even

higher immediate mortality than the generally accepted 50 per cent. for wounds penetrating the dura; but in the patients who recovered there would be a greater likelihood of freedom from subsequent complications. Careful follow-up records were kept; and as they covered a period of more than a year, they showed figures which might be termed end-results. There was not a single report of late hernia cerebri, meningitis or brain abscess. This was the most striking feature of the follow-up of the series of head wounds. Up to the present there had been two reports that suggested the existence of petit mal, but none of convulsive seizures. Turning to the symptoms that continued after the patients arrived in England or the various colonies, headache and dizziness were the most frequent complaints, either or both of these symptoms being reported in the first months after operation, and had persisted in 20 per cent. of the patients. There was, however, a general trend toward improvement. Other subjective symptoms, such as insomnia, hypersusceptibility to sounds, inability to concentrate, and so on, were not infrequently mentioned; but these had no proportionate relation to the gravity of the wound. The reports in but one case suggested the possibility that insanity might have developed. Gross paralysis occurring after wounds damaging the brain in the parietal and parietofrontal regions was comparatively infrequent, and the majority of paralyses showed improvement before the patients left the casualty clearing station. Subsequently the course of the paralyses or pareses ranged from progressive improvement to complete disappearance. The development of spasticity was reported in but one case, and there was not a single instance of persistent aphasia. The conclusion to be drawn from the follow up of this series was that the ultimate prognosis for patients with craniocerebral wounds after complete early operations was far more favorable and cheering than was generally thought to be the case. The total mortality for the operated head wounds, including deaths from other causes and deaths after leaving the hospital, was 42 per cent. in dural penetrations in the first series of cases, and was reduced to 29 per cent. in the second series of head wounds with dural penetration. The latter group of cases was treated at Mobile Hospital 2, A. E. F.

Dr. Neuhoef described the type of material that came under his care, the physical signs and neurologic manifestations, and gave in some detail the operative technic that was employed.

DISCUSSION

DR. FOSTER KENNEDY said that Dr. Neuhoef had covered such a wide field that it was difficult to decide which parts of his paper to discuss. At the beginning of the war the neurologists and the surgeons acting with them seemed to be very much in favor of very wide exploration in almost all cases of cranial injury. As the war went on, that policy was more and more modified, and it would seem that the modification was associated with good results. At the hospital to which he was first attached he saw more cases of brain abscess within three weeks than he had ever seen before. The policy which had been inaugurated there was that a man coming in with a cranial injury was subjected to a revision of the wound. In the course of that revision, if a crack of the cranium was found, a trephining operation was automatically done. The result of that policy was the production of innumerable cases of brain abscess. It was very difficult to get the surgeons to realize that though they were dealing with a fracture, it might be a fracture in good position. They had to acknowledge that if they were dealing with an infected fracture of the femur without displacement, they would not cut down on it and plate

it. But this procedure with the skull was followed because it always had been done as it was believed to be necessary. But it was not necessary. When those same men got in the habit of looking at these wounds, not for cranial injury but to see whether there was cerebral injury, the mortality was very markedly reduced. A rule was followed afterward that men coming in with head injuries were examined neurologically. If they had cerebral symptoms they were then subjected to cranial exploration. But whether they had a fracture or not, if they had no symptoms referable to compression they were left alone. As Dr. Neuhoef had said, the whole problem was sepsis. All these wounds were terrifically septic. The capacity of the scalp to become gangrenous and to have a widespread subcutaneous cellulitis of a fulminating type was a serious consideration. It was through such a surface that one had to operate. When a gratuitous opening was made in the skull, an avenue for infection of the meninges was laid open which usually had grave consequences and led rapidly to death. It was found that the dura had an ability to wall itself off, and the gross manipulations of the surgeons tended to break down those young adhesions. So they became very cautious and this caution was followed by success.

Dr Kennedy was glad to hear Dr. Neuhoef speak against the policy of digital manipulation of the brain. Sometimes digital manipulation failed to remove a bullet from below the surface of the brain, and yet the patient subsequently recovered. The capacity of the brain to encyst foreign bodies was remarkable, and even large pieces of metal seemed to be borne without much discomfort.

Dr. Neuhoef had said that he thought cerebral hernia was associated with a septic process. The speaker agreed with him up to a certain point, but he was not as pessimistic regarding the certainty of brain abscess following. The septic process was often quite superficial. Large hernias had disappeared under dressing every two or three hours; it was an edematous process which disappeared in four, five, six or seven weeks. Those patients, if transferred to England, would have died, for there had been a high mortality in patients taken back home; but when a patient could be kept in France he very often recovered. Those cases were most often the result of a purely local sepsis got rid of in the ordinary way by dressing very frequently.

There were some valuable lessons to be learned from the subsequent history of those cases. There never had been a time when so many men were so seriously hurt in the head as in the last few years. There had been ample opportunity to observe the results of very serious brain injuries in the mass. For instance, men returned to the firing line after they had been trephined a year or so before did not do well. They had all lost their nerve. They did not suffer locally from their injury, but they were not able to stand the strain, to adapt themselves again to their environment. But on the whole, very little after results, as far as ordinary peace time conditions were concerned, had developed. It was amazing that there were not more cases of epilepsy and more cases of mental breakdown as a result of head injuries. Emotional instability varied inversely with the seriousness of the wound; the more the man was wounded, the more calm his mind and emotional sphere.

Dr. Neuhoef had not said anything about filling in the open wound by the surgeon after the removal of missiles or operation for the relief of pressure—trying to do an Albee operation on the head by implantation of bone graft. In this operation there was always uncertainty whether or not the area would become septic again, and it was not advisable. The same operator would make an artificial skull by imposing a piece of metal in place of the bone

defect. That seemed to be very bad plastic surgery. It seemed neurologically wrong. If there was a well healed area with the individual not suffering from it, one ought not to interfere any more with it, and a metal cap could be given to be placed on the outside of the head—a solution unlikely to open avenues for bacteria.

Some of the worst cases Dr. Kennedy saw did remarkably well. He was amazed to discover the resistance of the brain to very severe injury. One man had had a very large glancing wound of the left frontal lobe with crushing depression fracture sustained forty-eight hours before. The wound smelled gassy. Fourteen pieces of bone were removed and some of the lining of the helmet, all from the brain tissue. The wound was left open, but all of the destroyed tissues of the scalp were cleaned away. The patient had a series of convulsions after the operation, became completely aphasic, recovered, and was sent to England, where he had a plastic operation done, and when last heard of he was doing perfectly well and working as a laborer.

Another man had a small cranial crack but was without symptoms other than headache and some vomiting. The wound was in the region of the left temporal lobe and he had been operated on but nothing was found. After operation he became motor aphasic. The wound was reopened and a subpial hemorrhage was found. The wound was widened and the hemorrhage left undisturbed. He subsequently made a complete recovery. There was a series of cases of men struck on the helmet and sustaining a very small wound, the whole force of the blow having been taken by the helmet. They came down concussed, with pulse below 40 and remaining between 40 and 50 for three days, during which time they would vomit. There was no sign of local brain pressure, but the spinal fluid came out under extreme tension. The symptoms would last from three to five days; they would be quite well for from three to five days, and then they would have a relapse and that situation might be maintained for eight weeks. Many such cases were seen. All these patients had the appearance of profound episodic compression of the medulla. By prolonged rest, without operation, they all recovered.

DR. GOODHART considered very interesting the fact related by Dr. Neuhof that he placed great reliance on the location of the injury through the neurologic reflexes, and thought that was very important. He had a case under observation in which deep seated neoplasm had been suspected, the only sign being absence of abdominal reflexes on the right side, and ten days later there was evidence of pyramidal tract derangement.

DR. NEUHOF thought the cases to which Dr. Kennedy referred were different from those seen in his own experience, that is, wounds in a state of infection. The treatment for them was different and consisted in not doing anything unless there were definite indications from a neurologic examination. At the base he saw six cases of hernia cerebri treated by persistent efforts at sterilization and which receded. Four patients developed subsequent complications and died anywhere from five to nine months after the recession of the hernia cerebri. This was surely ample reason for pessimism.

CLINICAL FEATURES ACCOMPANYING CHANGES IN THE SELLA TURCICA. Presented by DR. WALTER TIMME.

Dr. Timme considered it was difficult to announce, in these cases, which came first, the sella changes or the actual metabolic and blood disturbances. He did not wish to be understood as saying that the clinical manifestations were produced by these changes in the sella turcica. Thirdly, he was not talking particularly of the pituitary gland but of the changes in the sella turcica itself; and those two things were not synonymous.

The normal sella turcica in an adult was quite variable in size, the limits being fairly large, the average anteroposterior measurement being from 10 to 12 mm. and in depth 8 mm. The sella turcica in woman as well as the pituitary body was larger proportionately than in man. The anterior lobe of the pituitary body was vastly more supplied with blood vessels than was the posterior or the so-called middle lobe. If through some emotional or other disturbance the abdominal arterial supply was diminished as to capacity due to excess amounts of epinephrin or other sympathetic stimulant, such condition would be followed by an increased amount of blood in the pituitary body and would have its effect on the pituitary.

The manifestations produced by enlargement or disturbance of the anterior or posterior lobes occasioned a different symptomatology. Hyperplasia or stimulation of the anterior lobe produced genital enlargement and general bodily growth; and disturbance of the posterior lobe was followed by metabolic changes, such as increased or decreased sugar tolerance, blood pressure changes, and changes in tonicity of the smooth muscle fiber.

Granting most of these things, what happened to the sella turcica, the pituitary body in which was more or less affected by these conditions? With a sella turcica so small that the pituitary body fitted snugly in the cavity there was no allowance for much expansion, and any increase in size for any length of time was accompanied by certain pressure changes; so that headache in the region of a line drawn between the two temples was produced by a pituitary body enlarged beyond the point where the sella turcica would allow it to enlarge. If such a process continued for any length of time, certain changes took place in the sella.

The small sella turcica was not necessarily synonymous with a low activity of the gland. One might find associated with a small sella turcica a marked increase in growth. In some patients also there were marked symptoms of hyperpituitarism, prognathous jaw, spaced teeth and symptoms of acromegaly, and in addition disturbance of sugar metabolism, excessive growth of adipose tissue and similar conditions. Up to a certain point these conditions were progressive. If, however, there was too long continued increase in size or pressure of the pituitary body against its bony capsule, then things began to happen to the sella turcica. In the first place, the bony capsule began to enlarge, the pressure itself producing an enlargement in all diameters of the sella turcica; or the anterior or posterior wall or base became eroded and absorbed. For a long time it was believed that the change produced in such a capsule as the sella turcica would be indicative of the portion of the gland which was enlarged; if the anterior clinoids were eroded then it was concluded that the anterior lobe was the cause and sexual or growth changes were looked for; and if the posterior, changes in metabolism were expected. That was not always true; the clinical picture might be different, and the reason was that if there was an adenoma or a foreign body or hyperplasia of the gland, it did not necessarily transmit its pressure on the portion immediately surrounding it but might exert it on a portion quite distant which became eroded.

Another possibility was this: Realizing that the pituitary body compensated for certain activities of other glands, it was clear that the pituitary body itself must enlarge in the performance of this compensation.

There was such a thing as a small sella turcica and a closing in of the sella turcica. None had been seen at necropsy entirely closed in, but there were roentgenograms in which the anterior and posterior clinoid processes seemed to be united.

A large sella turcica, large as compared to the gland it contains, might be associated with manifestations of pituitary disturbance without a feature of disturbance appearing in the roentgen-ray picture.

DISCUSSION

DR. LEON T. LEWALD thought that Dr. Timme had given a most careful and conservative paper on the interpretation of the relationship between the pituitary manifestations and variations in the sella turcica. Since he had mentioned the heart, in regard to the compensation of the heart it might be apropos to refer to studies recently made in the army of the hearts of aviators under conditions of low pressure and low oxygen supply to determine if the heart dilated. Even in such a large organ, measuring from 12 to 13 cm. in diameter, it was difficult to differentiate a few millimeters in size as the aviator ascended 20,000 feet. Manifestly, then, still greater difficulty must be encountered in attempting to differentiate the slight changes in size of the pituitary fossa. In the aviator, account was taken of his size, weight and height. In the study of the sella turcica this was impracticable; for after considering a large number of healthy individuals, one would be struck with the extreme variations to be found in the pituitary fossa. Dr. Timme appreciated that fact and had stated definitely that under certain circumstances it was difficult to make the clinical manifestations jibe with no apparent change in the size of the sella turcica, and yet changes might be going on in the gland which did not cause any change in the bony formation. It was impossible to measure accurately the lateral diameter of the sella turcica, so the anteroposterior diameter and the vertical were the only two out of three dimensions usually considered. By stereoscopic roentgenographic examination a more accurate conception of the size of the fossa might be gained, and this should be made a part of the roentgen-ray examination. There were two things that were striking in Paget's disease: The conformation of the skull, which was almost the opposite of the conformation in cases of acromegaly; and the difference in the size of the frontal sinuses, which were greatly enlarged in acromegaly but in Paget's disease were very shallow. There was a prominence of the frontal bone which made one think there were large sinuses; but as a matter of fact they were exceedingly shallow. The syphilitic theory of bony changes in Paget's disease had been entirely exploded, though it was repeatedly diagnosed as syphilitic disease of the skull.

When a clinical history did not agree with the laboratory findings or roentgen-ray findings, it was well to trace the case up and identify the individual with the report of the special examinations.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, April 25, 1919

JAMES HENDRIE LLOYD, M.D., *President*

A CASE OF INJURY TO THE BRACHIAL PLEXUS, PROBABLY THE INNER CORD. Presented by DR. CHARLES S. POTTS.

History.—Lieut. J. was wounded on July 19, 1918, by a machine gun bullet which entered on the right side about two inches below the clavicle and three inches to the left of the shoulder joint. He at once lost power in the arm and hand, and for a short time spat blood. Complete paralysis of the arm with intense pain continued for ten weeks, after which the pain disappeared and

motion began to return. His condition improved for one month and then became stationary and remained so until he was first seen by Dr. Potts, about March 10, 1919.

Examination.—At this time examination revealed: Inability to flex the index finger at either the first or second inter-phalangeal joint, weakness in flexing the hand on the wrist and weakness of all the intrinsic muscles of the hand. In other words, only the muscles supplied by the median and ulnar nerves were affected. There was atrophy of these muscles and they presented a fully developed reaction of degeneration. Complete loss of pressure, tactile, pain and temperature senses were present on both dorsal and palmar surfaces in the middle, ring and little fingers and in the hand to a line running from the middle finger to about the middle of the wrist. Above this on the ulnar side of the forearm to the elbow there was diminished sensibility. At the present time there has been some improvement in motion, but practically none in sensation. This condition is very unusual. The syndrome resembles that described by Stewart and Evans and is due to injury of the inner cord of the brachial plexus. The symptoms do not indicate division of the inner cord, but indicate that it may be involved in adhesions and scar tissue.

PERIPHERAL NERVE INJURIES. MAJOR G. E. PRICE, MAJOR H. O. FEISS, and LIEUT. W. B. TERHUNE contributed the paper which was read by DR. FRANCIS X. DERCUM.

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DECEPTIVE HAND AND FOREARM MOVEMENTS IN PATIENTS WITH NERVE WOUNDS. Presented by MAJOR ANDREW H. WOODS.

Motor Symptoms.—Sensory changes seem more interesting to most examiners and there is a tendency to neglect motor symptoms and to overlook their significance. The important motor symptoms consist in diminution or loss of normal movements, addition of abnormal movements such as twitching and tremors, and substitution movements. In substitution movements the observer may be deceived by changes in position brought about by normal muscles or other forces which resemble the movements ordinarily due to muscles now paralyzed. Instances of these substitution movements cited were:

1. Elastic rebound of a joint due to the pull of soft tissues on the same side of a joint as the paralyzed muscles after they have been stretched by the active antagonists.

2. Movements produced by the tightening or relaxing of inert muscles through the action of normal muscles or other forces; for instance in patients with musculospiral paralysis the dead extensors of the digits hoist up the wrist into extension through the tension produced on them by flexing the digits.

3. The musculospiral extensors of the digits normally assist the interossei in extending the middle and distal phalanges, and they may replace the interossei in this movement when the ulnar nerve is paralyzed. Other "tricks" were mentioned by which lateral finger movements are imitated.

4. Pronation in cases of median nerve paralysis may be started by action of the musculospiral extensors and completed by gravity. Supination in cases of musculospiral paralysis may be effected by the biceps even with the elbow in complete extension. The trick of rolling the flexed hand into supination by allowing it to fall along the outer curve of the thigh was referred to as one that has caused mistakes in diagnosis.

ACUTE SOFTENING OF THE BRAIN AND CORD WITHOUT HEMORRHAGE IN NONPENETRATING GUNSHOT WOUNDS CAUSED BY IMPACT CONTUSION. Presented by DR. WILLIAMS B. CADWALADER.

Dr. Cadwalader said that his object in presenting the notes of the following cases seen in France was to call attention to the effect on the nervous system of the impact of a bullet or shell fragment without penetration. Softening of the brain and spinal cord can occur in nonpenetrating wounds without laceration or hemorrhage. The alterations of the central nervous tissue are caused solely by the local devitalization of tissue produced by the impact.

CASE 1.—This patient was first seen in an unconscious condition with left hemiplegia. There was a large depressed fracture in the right parietal region. The wound was nonpenetrating. Operation was performed and the depressed bone removed. It was discovered that the dura was not lacerated and no evidence of hemorrhage could be discovered. The patient died a few days later and at the postmortem examination it was found a large area of the right cerebral hemisphere was undergoing softening. No evidence of infection, hemorrhage or thrombosis could be detected with the naked eye.

CASE 2.—A soldier was wounded by a machine gun bullet, which lacerated the scalp and passed from the right to the left side, grazing the bones over the vertex of the skull. He became unconscious and suffered complete paraplegia. Repeated roentgen-ray examinations showed that there was no fracture. His paralysis improved slowly, and after six weeks he was walking with the assistance of a cane. Marked bilateral foot-drop persisted. The tendon reflexes were equal and only slightly exaggerated. A distinct Babinski sign was obtained but was not constant. The paralysis was flaccid and the gait was similar to that seen in peripheral neuritis. It is most probable that both paracentral lobules had suffered from the effects of the impact without hemorrhage. The absence of spasticity in the paralyzed muscle is singular. French writers have attempted to attribute this phenomenon to associated involvement of the cerebellum by *contre coup*, but this does not seem an adequate explanation. Dr. Cadwalader was inclined to believe that in cases of pure cortical paralysis there is always a tendency to flaccidity but it is not likely to be marked except in those cases in which only one segment of a limb is paralyzed.

CASE 3.—Severe intramedullary lesions of the spinal cord not uncommonly result from bullet wounds of the spinal column notwithstanding the fact that the spinal canal has not been penetrated and the dura mater has not been lacerated. An instance of this was found in a soldier who was hit in the back by a machine-gun bullet which was localized by the roentgen ray between the bodies of the third and fourth thoracic vertebrae. There was complete motor and sensory paralysis of all the parts below the fourth intercostal space. The patient finally died. At necropsy the dura was intact but the cord, at the level of the lesion, was swollen and soft. No microscopic examination was made, but to the naked eye, for the extent of at least one whole segment, there was necrotic softening without hemorrhage. Laceration and compression could be excluded.

The effects of contusion on peripheral nerves are very confusing. In examining patients a few days after receiving a wound of an extremity, it was frequently noticed that the extent of the paralysis was so much greater than would be expected judging, of course, by the location and character of the wound. Moreover, considerable return of function frequently took place in a few days.

The following case illustrates the condition.

CASE 4.—A soldier was hit by a machine-gun bullet in the lower jaw about half an inch to the left of the midline. The wound of exit was to the right of the midline posteriorly at the level of the third cervical vertebra. The spinal canal was probably not penetrated for the roentgen ray showed that the bodies of the vertebrae were not injured, but the course of the bullet must have been very close to the bodies of the third and fourth cervical vertebrae. The man stated that the instant he was hit he fell to the ground and that on arising, which he did without assistance, both arms were completely paralyzed. He said that he had not sufficient power in his arms to throw them over the shoulders of his companions who helped him to walk to the dressing station.

The next day there was complete flaccid paralysis of both upper limbs. The movement of shrugging the shoulders was not completely abolished but it was very weak and a little weaker on the left side. Cutaneous sensation was intact. The tendon reflexes could be obtained though greatly diminished on both sides. The lower extremities and other parts of the body were normal, but the tendon reflexes were exaggerated. The plantar reflexes were normal. After the first week motor power gradually began to return in the arms and at the end of two weeks the patient was able to extend and flex the forearm and fingers a little, but all movements were still very weak. The patient was finally transferred but was recovering rapidly.

It is not easy to determine exactly what the lesions were which had caused such profound paralysis, but it seems quite certain that the brachial plexus had escaped direct injury, because the course of the bullet was too high and it also appeared unlikely that the cord and its roots could have been destroyed. It is not improbable that contusion or concussion might have been responsible for at least part of such an extensive paralysis.

Mitchell, Moorehouse and Keen, in their book entitled, "Gunshot Wounds and Other Injuries of the Nerves," published in 1864, say on page 2: "A ball passing above the brachial plexus or just over any large nerve, will sometimes inflict injuries as severe and lasting with reference to the destruction of function, as the bullet which severs the nerve fibers themselves. For this reason we have been unable to classify nerve wounds so as to study the relation between the extent of nerve injury and the amount of the consequent shock."

NEUROPSYCHIATRIC PROBLEMS OF AN ARMY AT THE FRONT,
BOTH DURING AND IMMEDIATELY AFTER COMBAT was Dis-
cussed by MAJOR J. H. W. RHEIN.

Major Rhein said that the war neuroses as seen among the American forces presented some features which differed to a certain extent from those seen in the English and French armies. In the English army hysteria was more common in the soldier while anxiety states predominated among the officers. In the American army this was not true, anxiety states being common in the soldiers. The number of cases of major hysteria on the whole was less than in the French and English armies. Major hysteria was rarely seen at the front in army neurological hospitals where the cases seen could be classified for the most part as neurasthenics, psychasthenics, anxiety states, a few major hysterics and some which were examples of hyperemotivity representing what might be looked on as potential neuroses.

Soldiers who presented the symptoms of an excessive reaction to fear which for the moment incapacitated them and from which they recovered when removed from danger except for an anticipatory anxiety state, would develop fixed neuroses if they were not removed from exposure to front line experiences or if they were allowed to entertain prospects of returning to the front. The cases admitted to army neurological hospitals responded promptly to treatment of rest, good food, graduated exercises, suggestion and moral treatment. Between 60 and 70 per cent. of the cases were returned to their organizations within ten to fourteen days.

Fixed neuroses, consisting of major hysteria and anxiety states, were products of the contagion to which patients were exposed during their evacuation to the rear. In base hospitals were found hysterical manifestations in considerable numbers in contrast to the rarity of this condition at the front. The atmosphere at the front where these patients were treated within hearing of shell fire and where they understood that they were to return to their organization after some treatment, had a very different result from the standpoint of suggestion than that in the base hospitals which were far removed from the scene of warfare, where the safety of the situation emphasized in the minds of the men the dangers of the front, and where they picked up various symptoms from contact with other patients presenting fixed neuroses.

In over 50 per cent. of the cases coming into the army neurological hospitals the history contained a shelling experience. A diagnosis of hysteria was made in these cases. The trauma might be either emotional or physical. When the latter, the soldier stated that he had been blown over by a shell explosion. There was, however, no evidence on admission, of any actual cerebral concussion and rarely were the men unconscious when they reached the army neurological hospitals.

As to family predisposition in cases of war neuroses: A study of the histories of 332 cases showed that the family history was negative in 195 cases and positive in 137 cases for insanity, cancer, tuberculosis and nervous manifestations in parents, brothers and sisters. A history of previous nervous manifestations in the individual was found in about 50 per cent. of the cases.

Major Rhein said that there was no doubt that many healthy individuals developed in a short time due to the severity of their experiences, a state of nervous instability which would require months or years to develop in civil experience. Some cases showed psychotic reactions which presented transient symptoms suggestive of dementia praecox, manic-depressive psychoses and paranoid states.

A STUDY OF THE SPINAL FLUID IN CASES OF PRIMARY AND SECONDARY SYPHILIS. Presented by DR. JOSEPH McIVER.

This study was undertaken with the idea of determining so far as possible what percentage of cases of primary and secondary syphilis would show infection of the cerebrospinal fluid as might be demonstrated by the usual laboratory methods. The tests made on each specimen of spinal fluid were Wassermann test reaction, protein determination and cell count. Comparison of the blood Wassermann test has been made in every case with the findings in the spinal fluid. The duration of the disease, the present signs of syphilis and previous treatment have also been taken into consideration.

This study was made on a series of ninety-one cases, the majority of which were either in the primary or secondary stage of syphilis. The lesions manifested varied from early chancre to a fading secondary rash. A few of them,

however, were well past the secondary stage. Only one out of every four or five patients had taken any previous treatment and that was usually a very small amount.

All of these cases showed a 4+ Wassermann test reaction of the blood serum, as this was one of the determining factors as to whether or not a study would be made of the spinal fluid. The cell count was made immediately on removal of the spinal fluid. The average number of cells per cubic millimeter was 9. No effort was made to classify them. The greatest number of cells per cubic millimeter for any one specimen was 17. One of these was a case presenting a chancre of the lip with marked glandular involvement but no secondary rash; the others were cases of well-marked secondary syphilis. Taking a cell count of 5 per cubic millimeter as the average in normal spinal fluids, practically all of these cases showed a slight abnormality. Change in the spinal fluid of cases of secondary syphilis has been given from 10 to 90 per cent.

If we may consider a slight increase in cells an abnormality, then undoubtedly a large number of the primary and secondary cases of syphilis would show abnormal spinal fluid. This must be regarded as only of suggestive significance as there are a number of conditions that may produce an increase in cells. At the same time, we have to admit that cell counting is subject to error.

Only two of these cases showed a slight excess of protein in the cerebrospinal fluid, and curiously enough one of these was in the chancre stage, the other case was one of two years' duration with no previous treatment. The Wassermann test reaction of the spinal fluid was negative in every case with only two exceptions and they were of four and six years' duration respectively. Very little treatment had been given to either one.

In performing the Wassermann test reaction on the spinal fluid, 0.6 c.c. was used, 0.2 c.c. being the usual amount. The increased amount of fluid had no effect on the reaction.

Conclusions: 1. There is a slight increase of lymphocytes in the cerebrospinal fluid in the majority of cases of primary and secondary syphilis.

2. The increase in protein content does not appear as early as the increase in lymphocytes.

3. In this series, not a single 4+ Wassermann test reaction was obtained on the spinal fluid of primary and secondary cases of syphilis.

4. It does not seem reasonable to conclude that it can be determined by the examination of the cerebrospinal fluid from cases of florid syphilis just which cases will develop symptoms of the central nervous system.

DISCUSSION ON WAR NEUROSES

COL. SAMUEL LEOPOLD said it was impossible to discuss all the papers as his experiences were chiefly with the war neuroses. He saw very few head cases at the front. The same applied to cases of injury to the peripheral nerves. In his first station where his organization was brigaded with the French it was simply a case of evacuating the patient as soon as possible. In the next station there were better facilities for studying the war neuroses. Colonel Leopold had the opportunity of handling some 200 cases in this area; 50 per cent. recovered in less than a week and were sent to the front. Fright or terror with exhaustion constituted the clinical picture. There were some cases of hysteria. It was interesting to find in the hysterical cases that the symptoms could be removed quickly, though not so easily as in the fright cases. This led him to believe that the latter was possibly a potential or beginning neurosis.

MAJOR T. H. WEISENBURG said that there were three types of cases observed in the General Hospital in Plattsburg Barracks, the only war neuroses hospital conducted by the War Department in this country. The first type of cases were those who came from camps in this country; the second, patients who had been to France but who had not been in combat; the third, soldiers with actual fighting experience. There was some difference in the type of neuroses presented by these three groups. The first were chiefly hysterical, the second were made up of neurasthenics and anxiety groups, whereas the majority of the combat patients were so-called intractable hysterics, with the exception of the officers who presented anxiety types. There was not much difference in the hysterical manifestations between the overseas and the local troops, except that the former were perhaps more difficult to handle because of the many hospitals through which they had passed.

A very striking fact that impressed itself on an observer was the makeup of the war neurotic. Plattsburg is an old post in a town of about 10,000 people, and the patients and medical officers were intimately associated. Major Weisenburg was on duty in this hospital for a period of about four months. This is mentioned because when one lives among these patients their mental makeup is much more apparent than it is to anyone who sees them only occasionally. Many of the overseas patients wore decorations to which they were not entitled and which the War Department had not authorized. These men were boastful and more than willing to talk about their fighting experiences, which were usually imaginary. One rather expects this from soldiers but not to such a great extent. They were difficult to manage; that is, there were more complaints and more infractions of the rules of the hospital than is usual. Individually these men were not a likable lot. This was further impressed on Major Weisenburg, who, after this experience in Plattsburg, was ordered to Base Hospital No. 1 in New York City, where he organized and had charge of the organic neurological service and where most of the patients had peripheral nerve injuries. The contrast was more than striking, for in the organic group the boys were less boastful, less talkative, less complaining, easily managed and much more cheerful than the men of the neurotic type.

The curing of the neuroses in Plattsburg offered no special difficulty. Major Weisenburg had charge of all therapeutic measures and instituted a so-called "atmosphere of cure," similar to that adopted in other neurological centers abroad. All the medical officers, hospital corps men and nurses were given lectures on the nature and curability of hysteria. The patients were mentally boosted and with the teamwork thus instituted, practically all intractable cases were cleared up in about two months. Once the reputation of the hospital was established, the cure of even the most difficult hysteric did not take very long, the hospital corps men and nurses doing equally good work with the medical officers.

One very interesting observation was made in regard to the question of suggestion. Before the armistice, it was very easy to bring about various symptoms and many precautions were taken to guard against adverse suggestions. While the armistice made no appreciable difference, and there were no spontaneous cures, nevertheless it was almost impossible after the armistice to suggest new symptoms. As an example of this, Colonel deSchweinitz spent a week with these patients and did his utmost to suggest new phenomena in studying visual fields. His experience is interesting in the fact that it was impossible for him to accomplish this, although he found the usual tube fields and visual contractions of the ordinary hysteric.

Regarding peripheral nerve injuries: In organizing the service in Base Hospital No. 1, New York City, the usual faradic and galvanic batteries were discarded and the so-called condenser apparatus was used. This apparatus has a great advantage because it is possible to measure accurately the amount of current in microfarads necessary to produce stimulation of a nerve or muscle. A normal muscle or nerve will respond to about 0.03, whereas an interrupted nerve will not respond at all. A partially interrupted nerve will take about 0.1 microfarads. Also this current has the advantage in not being painful.

The greatest impression that the peripheral nerve cases made on Major Weisenburg was the ability of the remaining normal muscles to substitute for the loss of action of those which were paralyzed. For example, in musculospiral palsies, the patient by flexing his fingers, by means of the median and ulnar nerves, produced a pseudo-extension of the wrist, giving the appearance of extension in the musculospiral distribution. In cases in which the musculospiral nerve escaped, and in which the median and ulnar nerves were paralyzed, the opposite took place; that is, the patient first extended his wrist, causing an approximation of the fingers with the palmar surface, giving the impression of median or ulnar nerve action. The ability to do this depends largely on the person and on the length of time after the injury. A bright, active, ambitious boy, with thin fingers will be much more successful than a boy less perspicacious with thick fingers.

In this connection it is interesting to refer to a recent paper by J. R. Hunt, appearing in *Brain*, 1918, in which this author claimed that there are two systems of the motor fibers transmitted in the peripheral nerves. This author claimed that there are a certain number of nonmedullated fibers in peripheral nerves transmitting so-called paleokinetic fibers, subserving associated movements, which according to him, come back first in regenerating peripheral nerves. He based this theory largely on two cases of musculospiral palsy in which he had seen, what appears to Major Weisenburg to be nothing more than a pseudo-extension of the wrist, brought about by the action of the median ulnar groups. With Major Woods he had studied a great many cases of musculospiral palsy, in which all showed this phenomena. In a number of such cases, subsequent operation had shown the two ends of the musculospiral nerve to be completely divided and some distance apart. Dr. Hunt's theory therefore has no basis.

At first Major Weisenburg was somewhat loath to advise operations in peripheral nerve injuries unless there was definite evidence of total interruption. Experience, however, has shown him that wherever the progress is slow and not what it should be, an exploratory operation in the hands of a competent surgeon is advisable, for operation cannot do harm and in nearly all such instances, even when there is no actual interruption of a nerve, there has always been found sufficient surgical evidence to warrant operation.

LIEUT. GEORGE WILSON said that his neurological experiences in France were with Base Hospital No. 34 and with the Seventy-Eighth Division. During June, July and the early part of August, 1918, he saw a great number of cases of neuroses that had filtered all the way back to Base Hospital No. 34 which was located at Nantes, because the facilities for caring for these cases in the front area were not complete at that time. A great number of these patients responded readily to suggestion, isolation and other methods so that only a few had to be sent to Base Hospital No. 117. At Nantes Lieutenant Wilson saw a limited number of peripheral nerve cases, but as

Base Hospital No. 34 was the only functioning surgical hospital in that area, those cases were evacuated to other hospitals not entirely completed in order to make room for the great number of wounded who came in during those critical days.

About the middle of August, Lieutenant Wilson was assigned to the Seventy-Eighth Division as psychiatrist and remained with that organization until November 25. When the soldiers suffering from the various neuroses were seen in the triage and kept, so far as possible, in a nearby field hospital, 75 per cent. were returned to their organizations in ten days. When these men were seen before their tremor and other symptoms had become fixed it was a relatively simple matter to straighten them out and return them to duty, much easier than it had been in the base hospital. In the last three weeks of the war, which Lieutenant Wilson spent in the Argonne sector, the number of cases of the psychoneuroses that he saw were very few. This may have been due to the fact that the weak sisters had been weeded out or, what is more likely, that the outlook had changed from a long and cruel war to a short and merry one.

COL. WAYNE BABCOCK stated that he had been especially interested in cases of nerve injury. Over 350 had been admitted to General Hospital No. 6, Ft. McPherson, Ga., and something over 200 patients had been operated on. About 260 nerves had been explored under the knife. Something less than half of the nerves had been subjected to neurolysis and the rest to suture. The interesting feature had been that the proportion of sutured cases had increased with experience. This applied especially to certain neuromas in continuity with physiologic interruption, the paralysis from which had persisted in a number of cases without improvement three or four months after neurolysis and hersage. The sutured cases in general were giving more promise. It was too early to say more. There had been a number of interesting phases in the work; one was the study of partial nerve palsies in relation to intravenous localization. For example: In a case recently operated for foreign body near the internal condyle of the humerus the ulnar paralysis was limited to the flexor carpi ulnaris with complete sensory loss. As the sensory fasciculi and those supplying the flexor carpi ulnaris run in the anterior inner portion of the ulnar nerve at the elbow, it was interesting to see whether that corresponded to the lesion when the nerve was exposed. On exposure a little pocket was found in the anterior internal portion of the nerve, in which rested a small foreign body. In some of the other cases, there had been a somewhat similar correspondence between the tracts interrupted and the areas paralyzed. Most interesting had been the mapping of nerve patterns of exposed nerve trunks by the electric current.

Some of the results of operation had been rather anomalous until explained by careful study of the cases. For example: In one case of peroneal division, for which a suture was done, the extensive sensory loss apparently cleared up promptly. The boy said he had return of feeling on the third day after operation, and it was found that he had practically regained all sensory power, both to touch and to pain. The explanation was found in the fact that the musculocutaneous branch had not been divided by the injury and had been freed at operation, while only the anterior tibial fasciculi had been sutured. Of course in this case there was no early return in motion. Another interesting phase has been the size of the gaps that could be bridged, a technical problem of importance owing to the unreliability of nerve grafting. By adopting various expedients, in about 120 cases with gaps from 3 to

15 cm. in length, no case was found in which an end to end suture could not be made. The gaps were overcome (1) by the displacement of the nerve from its normal location—as the ulnar to the front of the elbow; the median from a deep to a superficial location; (2) by flexion or extension of adjacent joints; (3) position, adduction of arm, adding 5 to 8 cm. of slack to the cords of the brachial plexus; (4) by the normal slack and elasticity of nerve trunks.

To this has been added the interesting problem that has appealed to many, as to whether the nerve could be lengthened progressively under multiple operations. This Colonel Babcock had a chance to observe in one case in which a man had an injury extending from the chest along the arm. There was an extensive laceration of the median and ulnar nerves. In the first operation, a gap of about 5 cm. of the median nerve and 11 cm. of the ulnar nerve were overcome with suture. The nerves had so elongated three months later, that it was possible to excise an additional 10 cm. from the median nerve and 6 cm. from the ulnar nerve, and obtain end to end apposition with suture, although a total of 15 and 17 cm., respectively, had been removed from the nerves.

The rapidity of healing in nerves was also brought out by another case in which there was a gap of about 12 cm. in the sciatic nerve. During this operation, despite an acute flexion of the leg on the thigh, the tension was so great that when the man moved a little on the table after four or five stitches were introduced, the nerve pulled apart. Colonel Babcock went ahead again, and the nerve was finally well sutured by fine silk sutures. The patient was tied up, as they thought, very carefully, but that night, as Colonel Babcock went to the ward, he found the man had flexed the thigh on the abdomen and knew the nerve was pulled apart. Twenty hours after operation Colonel Babcock reoperated, but, on exposing the nerve, it was found to be absolutely together, and on taking hold of the ankle and extending the leg twenty degrees, he could see not the slightest tension on the sutures. The explanation was that the hamstrings had quickly adhered to the nerve, splinting it perfectly. As a result, instead of waiting many weeks to extend flexed joints, Colonel Babcock now started them quite early.

An added reason came from observing other men who deliberately extended their arms or legs, although they had been told not to do so, and who, despite this, had evidence of returning function corresponding well with the progress made by patients who had had short gaps overcome with suture.

MAJOR K. W. NEY stated that during the American offensive he was associated with evacuation hospitals or mobile units near the front in the capacity of senior member to a neurological operating team. This team did head surgery almost exclusively. In all cases as complete a neurological examination was made as circumstances would permit. All brain operations were done under local anesthesia, which proved to be the anesthetic of choice. When head wounds were complicated by other injuries the head operation was done under local anesthesia and a general anesthetic given for the debridement of other wounds. The operative technic employed was complete excision of the scalp wound; removal of the bone injury *en bloc*; evacuation of the disorganized brain substance by having the patient blow against his closed lips, or by coughing, the increase of intracranial pressure by this method proved very effective in the removal of the destroyed, oozing, semifluid brain substance. The dura was sutured whenever possible, but in all cases the cranial defect was covered by scalp, and drainage was never used.

If the patient survives the anatomic death which usually takes place in the first few hours, he then has to face the dangers which so often resulted in an infective death. It was in the prevention of this infective death to which we directed most of our efforts and where we found our reward by doing an early and complete operation. The mortality early in the service in gunshot injuries of the brain was between 50 and 60 per cent. in patients who had avoided an anatomic death, but who died from infections. By the above operative technic, advocated by Colonel Cushing, we were able to cut this figure down to less than 10 per cent. In a series of seventy-nine cases Major Ney's team lost but five from all causes. All brain cases were retained for a period of from eight to ten days after operation and it was often remarkable to watch the clearing up of preoperative neurological symptoms during this period. The most remarkable experience, however, has been the clearing up of many cases which looked hopeless at the time of operation, as well as when they were evacuated back to the base. The prevention of the infective sequela promised them life, but it seemed almost as though it would be better to let them die than to prolong an existence of absolute helplessness—but the function of the members of the team was to save life, and in doing this thought they had done all; but what nature and reeducation has done in many of these cases has been most remarkable. In a recent visit to Cape May, N. J., Major Ney found a patient whom he had classified in his personal records as dead. This man had had an extensive amount of brain destruction with a complete right hemiplegia and aphasia. At operation a foreign body was removed from the lateral ventricle and he was not expected to live. The man was evacuated and it was thought that he had died. You can imagine Major Ney's surprise when he found that soldier playing ball and talking perfectly.

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No. 3

PERIPHERAL NERVE INJURIES CONCOMITANT TO GUNSHOT WOUNDS

PRELIMINARY REPORT ON THE DIAGNOSIS, OPERATIONS,
PROGRESS AND RESULT OF TREATMENT IN
FIVE HUNDRED AND TEN CASES

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FORT MC PHERSON, GA.

Four hundred and nineteen patients with peripheral nerve injuries were admitted to U. S. General Hospital No. 6, between October, 1918, and May 1, 1919. Table 1 shows the actual number of each individual nerve that was injured, the operations which were performed on that nerve, and the number which up to May 1 had not been operated on. Table 2, independent of time, shows the number of the individual nerves that showed no improvement, the number that were improved or cured, as the result of operation and treatment or treatment alone, and the number that have not, since their admission to the hospital, returned for a second examination. Table 3 shows the actual number of nerves that were made worse, remained the same, were improved, much improved and the number that were cured; also the operations that brought about these results.

Most of the patients entered the hospital at least four months after the infliction of the wound, and a large majority were not operated on until at least six months after sustaining their injury. All patients, with one or two exceptions, were examined at least once before the operation. Operation was advised only in those cases in which the neurological examination justified the diagnosis of complete physiologic block. We considered physiologic block to be present when there existed total paralysis with atrophy of the affected muscles, complete reaction of degeneration, and in a mixed nerve loss of sensation.

TABLE 1.—NERVE OPERATIONS PERFORMED AND NUMBER NOT OPERATED ON IN U. S. ARMY GENERAL HOSPITAL No. 6, FORT MCPHERSON, GA.

	Operations				Nonoperative or Not Examined Since Operation	Total
	Neurolysis	Hersage	Suture	Total		
7th.....	2	2	..	2
S. A.	1	1	..	1
C. P.	2	2	2	4
B. P.	3	6	2	11	15	26
C.	1	1	1	2
M. C.	3	4	3	10	6	16
M. S.	8	12	17	37	48	85
H.	8	15	43	66	37	103
M.	12	14	16	42	38	75
I. C.	1	..	4	5	6	11
R.	3	..	2	5	6	11
A. I.	1	1	2	1	3
P. I.	1	1	..	2	2	4
L. P.	1	1
S.	1	16	19	36	38	74
A. C.	1	1	7	8
E. P.	2	1	11	14	23	37
I. P.	2	2	4	7	11
I. S.	2	2	5	7
A. T.	3	8	11	3	14
P. T.	2	..	2	4	11	15
M. C.	1	1	..	1
Total.....	48	75	136	259	252	511

TABLE 2.—NUMBER OF PATIENTS THAT SHOWED IMPROVEMENT, NUMBER IMPROVED OR CURED AND NUMBER THAT DID NOT RETURN FOR EXAMINATION

	Operative					Nonoperative				
	Worse	Same	Improved	Much Improved	Cured	Same	Improved	Much Improved	Cured	Not Returning for Examination
7th.....	..	2
S. A.	1
C. P.	1	2
B. P.	1	8	3	6	5	1	3
C.	1	1	..
M. C.	4	1	4	1	1	2	..	1	2
M. S.	15	11	7	4	9	5	2	9	23
U.	1	33	24	5	3	6	6	2	4	19
M.	12	21	5	4	1	10	2	4	16
I. C.	4	1	..	2	2	1	3
R.	1	2	..	4	3	1	..	1	1
A. I.	1	1	..	1
P. I.	1	1	2
L. P.	1
S.	26	9	1	..	8	7	1	2	20
A. C.	1	1	6
E. P.	11	2	..	1	7	3	2	1	10
I. P.	2	1	1	2	1	..	4
I. S.	2	2	1	2
A. T.	1	2	2	1	1	..	1	1
P. T.	2	2	6	2	..	3	110
M. C.	1
Total.....	2	125	86	29	16	47	52	15	28	220
258					142					

TABLE 3.—OPERATIONS PERFORMED ON NERVES AND RESULTS

	Worse	Same	Improved	Much Improved	Cured	Total
Neurolysis.....	0	10	22	7	9	48
Hersage.....	1	18	39	12	7	75
Suture.....	0	100	31	4	1	136
Totals.....	1	126	92	23	17	259

We do not believe it is possible to foretell, even in the presence of symptoms of complete physiologic block, whether this was due to an interruption of continuity, or to the presence of scar tissue in a nerve. Operation was also advised in those cases that showed marked irritative phenomena, such as constant pain and very marked vasomotor and trophic disturbance in the absence of vascular involvement. In some cases there existed a doubt as to the necessity of operation, and these cases were reexamined in from two to six weeks before final advice was given.

A group of cases was examined in which loss of function was the prominent symptom; however, the motor disturbance was not due to neurological involvement, but was caused by injury to muscle, bone or joints. These cases were referred back to the surgical department with the diagnosis: "neurological examination negative."

There are two points of view from which the improvement and cure of a patient can be considered:

1. A patient may be considered improved, or cured, who has a beginning return of function, or almost complete return of function as the result of the education and increased power of adjuvant muscles, and of learning new ways in which to bring about the necessary movements of his extremities, with the recurrence of sufficient sensation to prevent the unconscious infliction of injury.

2. From the point of view of neurology, a patient is not considered cured until there has been reestablished to almost a normal degree the functions of sensation and motion in the region supplied by the affected nerve or nerves.

We have, in arriving at our conclusions, very closely followed the latter point of view, and consequently the improvements, marked improvement and cures are rather conservative estimates of the actual functional condition of the patients.

In practically all of the cases the date of injury anteceded the first examination, or operation, by at least five months, thereby giving a reasonable time for the appearance of some clinical signs of regeneration of the affected nerves.

The study of these cases shows that 511 nerves were affected up to the time this report was written. Of this number 259 were operated on, this number being exclusive of those who have not returned for a second examination. Two hundred and fifty-two were not operated on, or if operated on, have not returned for examination. Of the 252 nonoperative cases, 142 have been examined two or more times, 110 of the patients not having returned for a second examination. The actual results are therefore based on 400 injured peripheral

nerves which have been examined, with few exceptions, at varying intervals of from two to six weeks after the patients entered the hospital.

Of the 259 cases, forty-eight were neurolyzed, seventy-five were hersaged, and 136 were sutured. By neurolysis we mean that the nerve was freed from surrounding inflammatory tissue, and in a few cases the nerve sheath split. Hersage is the combing, or fiber disassociation, performed on a nerve by making a number of incisions with a very sharp knife in the longitudinal axis of the nerve. Suture is the approximation of two ends of a nerve after resection to normal fasciculi, and then with proper material sewing through the sheath, bringing the ends accurately together.

Table 1 shows the actual number of times that each nerve was involved. In our series of cases the ulnar nerve predominates, being affected 103 times; next in frequency comes the musculospiral, 85 times; then the median and sciatic, 75 and 74, respectively; then the external popliteal 37, and the brachial plexus, 26 times.

For purposes of study in this report we are going to eliminate the 110 nerves which have not had second examinations. Many of these patients are in the hospital; some of them have been operated on during the month of May, and will appear in later reports, thereby leaving 400 cases as a basis.

Of these 400 cases, operations were performed in 259, showing 65 per cent. of cases operative, and 35 per cent. nonoperative. Of this number 18 per cent. were neurolyzed, 28 per cent. were hersaged and 54 per cent. were sutured. I am rather inclined to believe that as these cases are observed for a longer period the percentage of operations will be somewhat increased.

Table 3 shows the actual number of nerves after neurolysis, hersage and suture that remained the same or were improved, much improved or cured. Of all operative cases, independent of the operation, about 50 per cent. remained the same; 35 per cent. were improved, 9 per cent. were much improved and 6 per cent. were cured. After neurolysis 20 per cent. remained the same; 45 per cent. were improved; 15 per cent. were much improved, and 20 per cent. were cured. After hersage 20 per cent. remained the same, 54 per cent. were improved, 16 per cent. were much improved, and 10 per cent. were cured. After suture 25 per cent. were improved, less than 1 per cent. were cured and the balance remained the same.

After neurolysis, more than 33 per cent. of the patients were markedly improved in from four to eight weeks; in less than twenty-four weeks nine were completely cured, some patients being cured as early as the fifth week. After hersage, at the end of twelve weeks,

25 per cent. of the patients were markedly improved, and in less than twenty-four weeks seven were cured. After suture, there were comparatively few patients that showed any improvement under sixteen weeks; after this time quite a number of them began to show improvement, especially in sensation; this is to be expected. There has been a marked improvement in the general appearance of the affected part, and in the comfort and mental attitude of the patients, and as time goes by we confidently anticipate a high percentage of cures in these cases.

PROGRESS IN OPERATIVE AND NONOPERATIVE CASES

In the operative cases, independent of operation, 44 per cent. of the cases were improved and 6 per cent. were cured; in the nonoperative cases 50 per cent. were improved and 17 per cent. were cured. If we exclude the suture cases, in which as yet it is too soon to expect much improvement, there remain 122 cases in which either herbage or neurolysis was performed. Of these cases twenty-six, or 21 per cent., remained the same; sixty-one, or 50 per cent., were improved; nineteen, or 16 per cent., were much improved, and sixteen, or 13 per cent., were cured. There were 142 cases in which no operation on the nerve was performed, and which were examined two or more times; in this group we find that forty-seven, or 33 per cent., remained the same; fifty-two, or 40 per cent., were improved; fifteen, or 10 per cent., were much improved, and twenty-eight, or 17 per cent., were cured.

On comparing the percentages in the two latter groups of cases, and taking into consideration the fact that the nonoperative cases were milder cases, and all gave a history of progressive clinical improvement, and that the operative cases were the more severe cases, and all gave a history of cessation of improvement for several months (and this in most cases was confirmed by two or more examinations), and that the large majority of these cases were examined from one to three months after operation, we find that 33 per cent. of the nonoperative cases remained the same as compared with 21 per cent. of the operative cases which remained the same; that 40 per cent. of the nonoperative cases were improved as compared with 49 per cent. of the operative cases; that 10 per cent. of the nonoperative cases were much improved as compared with 15 per cent. of the operative cases; and that 17 per cent. of the nonoperative cases were cured as compared with 13 per cent. of the operative cases.

These percentages, deduced from almost an equal number of operative and nonoperative cases in all of which the patients received their wounds in the same way, more or less about the same time, all subjected to similar early treatment and like treatment after arriving at the hospital, show first that operation in these cases, instead of doing

harm, hastens and makes more certain the improvement; in fact, we believe that if these 122 patients had not been operated on and had been treated in the same manner as the nonoperative cases, few, if any of them, would have shown very much improvement. A few patients were temporarily rendered worse after operation. This applies especially to the neurolyzed and hersaged cases; but almost without exception improvement followed and has continued. After hersage, in some cases improvement was delayed for a considerable time, and was then followed by a rather rapid disappearance of symptoms.

Disturbance of motion, sensation, pain and vasomotor and trophic manifestations did not become ameliorated or disappear simultaneously after the operation. In nearly all cases, pain was promptly relieved. In only a very few did pain persist, or occur in the distribution of the affected nerve after the operation. In no case of our series has the pain persisted for any length of time after operation. The vasomotor and trophic manifestations, in absence of vascular complications, in some cases were markedly improved, and in many cases disappeared after operations. There are, however a few in our series in which these disturbances have not been relieved by operation.

After neurolysis there is usually a prompt improvement in the sensory and motor disturbances.

After hersage there may be an improvement in motion or sensation, the two not necessarily improving, or returning at the same rate.

After suture the improvement that has been noted is return of the protopathic sensation, and progression toward the periphery of the point at which deep percussion tingling manifests itself.

Brachial Plexus Injury.—From a study of our tables, we find that there is a very marked tendency for injuries of the brachial plexus to be followed by a comparatively good functional restoration of the upper extremity, independent of whether they are operated on or not. We have had twenty-six cases of brachial plexus injury; of that number twenty-three have been examined two or more times; eleven were operated on, and twelve were not operated on. The operative cases all showed improvement, as did the nonoperative cases, although the manifestations of the operative cases were much more severe than of the nonoperative cases, and, according to the history, had ceased to improve for several months; however, following operation, improvement promptly occurred.

Median Nerve Injury.—The median nerve was affected in seventy-five instances; of that number fifty-nine were examined two or more times, forty-two were operated on, and seventeen were nonoperative. We noticed in this nerve that pain was rather a common symptom; vasomotor and trophic disturbances were frequently present, and when

the nerve was severed, the absolute loss of sensation was limited to the palmar surface of the two terminal phalanges of the index and middle fingers and dorsal surface of terminal phalanges of index and middle fingers; that on the palmar surface of the hand and proximal phalanges of the index and middle fingers, and the palmar surface of the thumb, tactile sensation was frequently lost, but pain stimulation gave rise to delayed, radiating, burning sensations. In those patients that were recovering, the muscles of the forearms supplied by the median nerve were the first to regain their function, with the possible exception of the flexor longus indicis. We notice in several cases that this muscle seemed to lag behind the other flexors in regaining its function. The thenar group of muscles was very slow in recovery, with the exception of the mild cases; tactile sensation was very long in reappearing in the terminal phalanges of the index and middle fingers.

Musculospiral Nerve Injury.—The musculospiral nerve, which was involved in eighty-five instances, operated on in thirty-seven cases, nonoperative in twenty-five cases and not returning for examination in twenty-three cases, shows a very marked tendency to recovery. This is evidenced in both operative and nonoperative cases. The sensory disturbances, even in instances of complete severance, are very limited, in most cases being limited to the dorsal surface of the proximal phalanx of the thumb and the small area on the dorsum of the hand at the base of the index and middle fingers. If this nerve is injured high up in the arm, or even in the axilla, the long head of the triceps may escape; when it is injured low down near the elbow, the entire triceps escapes. If the nerve is injured close to the elbow, the supinator longus is usually not involved. When the improvement takes place, the extensors of the wrist always recover before the extensors of the fingers.

Ulnar Nerve Injury.—The ulnar nerve was involved in 103 instances; of that number, sixty-six were operated on, eighteen were not operated on, and nineteen did not return for a second examination. Improvement in the ulnar nerve seems to be slower than in most of the other nerves; and, in fact, in those cases in which the nerve is very severely injured, although sensation and the function of the muscles of the forearm supplied by the ulnar nerve return in the expected time, the wasting and the power of the muscles of the hand supplied by this nerve does not seem to improve. In nearly all of our cases in which the ulnar nerve has been severely injured there has been very little improvement in the volume, tone and power of the muscles of the hand supplied by this nerve; in many cases, however, the functional result has been good.

We have noticed nothing very unusual in following our cases of sciatic, external or internal popliteal injuries, with the possible exception of the facts that vasomotor and trophic disturbances are more apt to occur in the distribution of the external popliteal than the internal popliteal; that these disturbances rather disappeared after operation; that recovery in the domain of the external popliteal is usually more complete than that in the internal popliteal; that in injuries of the sciatic nerve, the fasciculi which go to the external popliteal are more apt to be injured than those that go to the internal popliteal, and that in those cases in which causalgia occurs following injury of the sciatic nerve, the pain usually occurs in the distribution of the internal popliteal.

CONCLUSIONS

1. Careful neurological examinations should be made of each patient. When this examination justifies the diagnosis of complete physiologic block, taken with a history of cessation of improvement for from four to eight weeks, operation is indicated.
2. The presence of persistent pain or marked vasomotor and secretory disturbances, with a history of cessation of improvement for some time, calls for operative interference.
3. Following operation as soon as the surgical condition permits, energetic physiotherapy should be instituted; this should consist of passive massage; active exercise; electrotherapy, the current which will produce muscular contraction being used; proper splinting to prevent deformity; overstretching of paralyzed muscles, or contraction of paralyzed muscles, while still permitting as much freedom in motion of the affected part as possible.
4. The patient should be encouraged to cultivate a tranquil, optimistic attitude toward his disability, and to cooperate in his treatment.
5. From the neurological examinations, supported by subsequent operative findings, it is our opinion that from 60 to 75 per cent. of the patients at this hospital who have nerve injuries would not recover lost function without operative interference.
6. The operative cases have not been under observation a sufficient length of time to arrive at a more than tentative conclusion as to ultimate outcome.
7. The neurolysis and hersage cases have shown a considerable number of improvements, which we believe will be increased at a later date. Although measurable, objective improvement in the suture cases has been very limited, it is yet too early to expect any other result.

BULBAR PARALYSIS OR AMYOTROPHIC LATERAL SCLEROSIS?

A CLINICO-PATHOLOGIC NOTE *

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After Duchenne's first description of progressive bulbar paralysis¹ this disease was generally looked on as an independent clinical and pathologic entity. Later, numerous observations showed that it may form a part of the clinical pictures of amyotrophic lateral sclerosis, of chronic anterior poliomyelitis (subacute general anterior spinal paralysis of Duchenne) and progressive muscular atrophy (type Aran-Duchenne). The symptoms and signs are sometimes so confusing that a differential diagnosis between these four types is very difficult, and some authors (Starr,² Gowers³) have come to look on at least some of these conditions as practically identical diseases. The following clinico-pathologic report tends to demonstrate the identity of amyotrophic lateral sclerosis and chronic bulbar paralysis.

REPORT OF A CASE

Mrs. E. R., 62 years old, admitted on Dec. 13, 1918, to the county hospital, service of Dr. Frederick Tice, complaining of difficulty in swallowing, talking and walking, about one and a half years previously had noticed weakness of the upper and lower extremities and swelling of the latter. The weakness progressed and at the time of admission she could neither walk without support nor pick up small objects. During the past year her voice had become hoarse, "stumbling," her speech unintelligible, and for the last two months she had had dysphagia, especially for liquids.

Previous History.—She had had asthma since childhood, diphtheria at the age of 8. Syphilis and other constitutional diseases were denied. She had amenorrhea at the age of 36.

Family History.—Her father and mother died of old age.

Examination.—The patient sat upright in bed with the head well forward and her back markedly bowed anteriorly, constantly coughing and with a

*From the pathology laboratories of Cook County and Psychopathic Hospitals, Chicago.

1. Duchenne (de Boulogne): Paralyse progressive de la langue, du voile du palais et des lèvres, Arch. gén. de méd. 2:283, 431, 1860.

2. Starr, M. A.: Organic and Functional Nervous Diseases, Ed. 3, Philadelphia, Lea & Febiger, 1909, p. 280.

3. Gowers, W. R.: Diseases of the Nervous System, Philadelphia, P. Blakiston's Son & Co., 1892, Vol. 1.

peculiar facial expression. The muscles of the lower half of the face were drawn, retracting the corners of the mouth which was slightly open, the frontal muscles contracted, saliva constantly drooling. She could wrinkle the forehead, close the eyes and move them in every direction. The muscles of mastication and of lips, chin and tongue were markedly atrophied. The tongue could not be protruded beyond the teeth; it was thin, flabby, corrugated and showed marked fibrillary twitchings. The uvula and soft palate were motionless and did not respond to irritations; they appeared atrophied. The neck was thin, not rigid, the shoulders high, held as in forced breathing. The lungs revealed numerous high-pitched piping sounds typical of asthma. The heart was slightly enlarged, but showed no murmurs. The abdomen was negative.

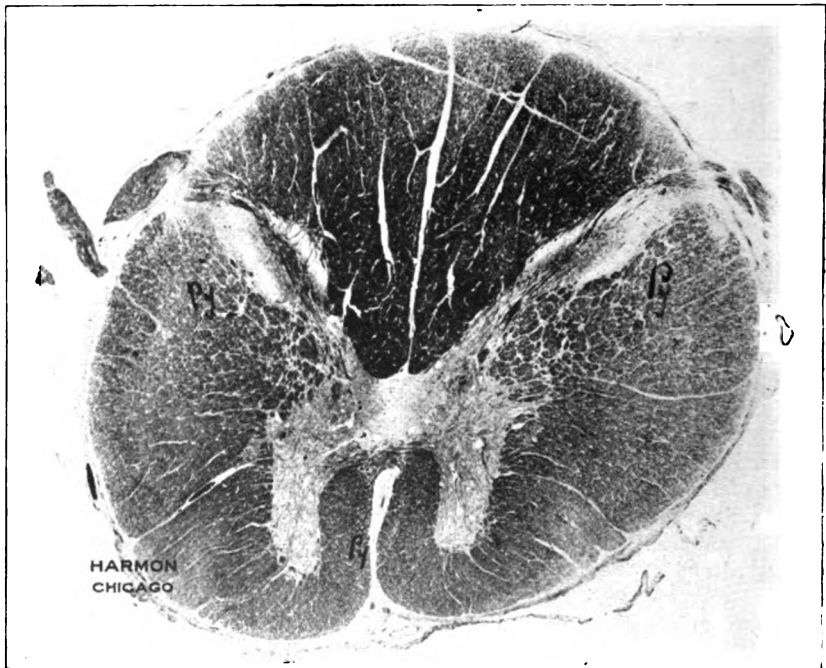


Fig. 1.—Second cervical segment. Posterior roots cross the posterior horns and are well developed, in contrast to the anterior horns, which appear smaller than normal and without any anterior root fibers visible. Fibers of the eleventh nerve, normally crossing the lateral columns, are also absent. The posterior, the lateral and anterior columns, including the pyramidal tracts (*Py*), do not show any visible or marked changes. Weigert-Pal stain, $\times 9$.

Extremities.—Active and passive movements were possible, without rigidity, spasticity or deformities. The feet were drooping; small muscles of the hands, the biceps, deltoid, and calf muscles were somewhat atrophied, especially the hand muscles, and fibrillary twitchings were quite marked in the deltoid and biceps of both sides. Reflexes: Pupillary, normal; triceps, knee, ankle and jaw jerks were all exaggerated, especially on the right; no clonus. Oppenheim's sign and the Chaddock and Babinski reflexes were absent. The voice was hoarse,

hardly audible; speech unintelligible, anarthric; mentality, good. Urine contained albumin, hyaline and granular casts. No serologic tests were recorded.

The anarthria, dysphagia and respiratory difficulties progressed and on Jan. 10, 1919, the patient died

Necropsy Examination.—The necropsy three days later, revealed emphysema and edema of the lungs, hypostatic hyperemia of the liver, diffuse nephritis, disseminated petechial hemorrhages in the gastric mucosa, moderate senile sclerosis of the aorta, marked atrophy of the interossei muscles of both hands, extensive fibrous peritonitis; the cerebral pia congested, transparent, not thickened and not adherent to the brain; normal sulci, convolutions and ventricles; the seventh, ninth and twelfth cranial nerves were visibly atrophied.

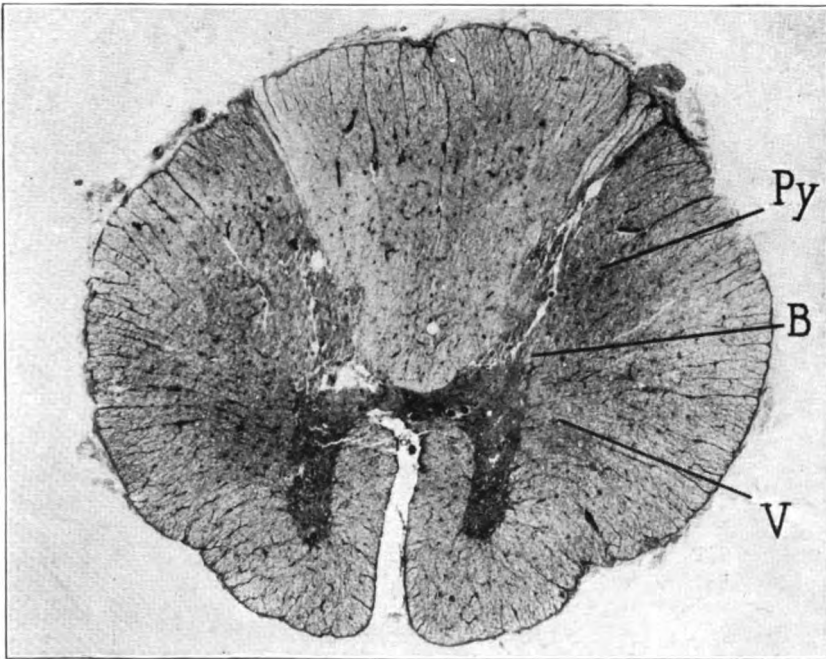


Fig. 2.—Tenth dorsal segment. The posterior and anterior columns are normal, showing, especially the posterior columns, a great number of vessels. In the lateral columns, the tracts of Flechsig and Gowers are also normal, while the areas of the crossed pyramidal tracts and the limiting lateral layer, its ventral portion, are degenerated, appearing darker stained than the normal portions of the spinal cord. *Py*, crossed pyramidal tracts; *B*, dorsal part of the lateral limiting layer, known as Bechterew's medial bundle of the lateral columns, is not degenerated; *V*, ventral part of the lateral limiting layer shows marked degeneration. Alzheimer-Mann stain; frozen section; $\times 13$.

Sections of the medulla, cervical and upper portions of the dorsal cord stained with Weigert-Pal showed atrophy of the nuclei of the twelfth, ninth, tenth and seventh cranial nerves and the fifth motor nerve and the corresponding nerve roots with practically normal white tracts in medulla and cord. The pyramidal fibers, as the photomicrograph (Fig. 1) shows, were normal in

amount though they appear less stained than those of the posterior columns. Both white and gray matter showed numerous congested, thickened and slightly infiltrated vessels (Figs. 2 and 3). The same excessive vascularization was present in the pia which was markedly infiltrated with lymphocytes (Fig. 3) Longitudinal sections of the pyramidal tract, stained with Mallory-Jacob (fuchsin and anilin blue), Alzheimer-Mann (methyl-blue eosin), and, still better, with Marchi or Bielchowsky and counterstained with the former methods

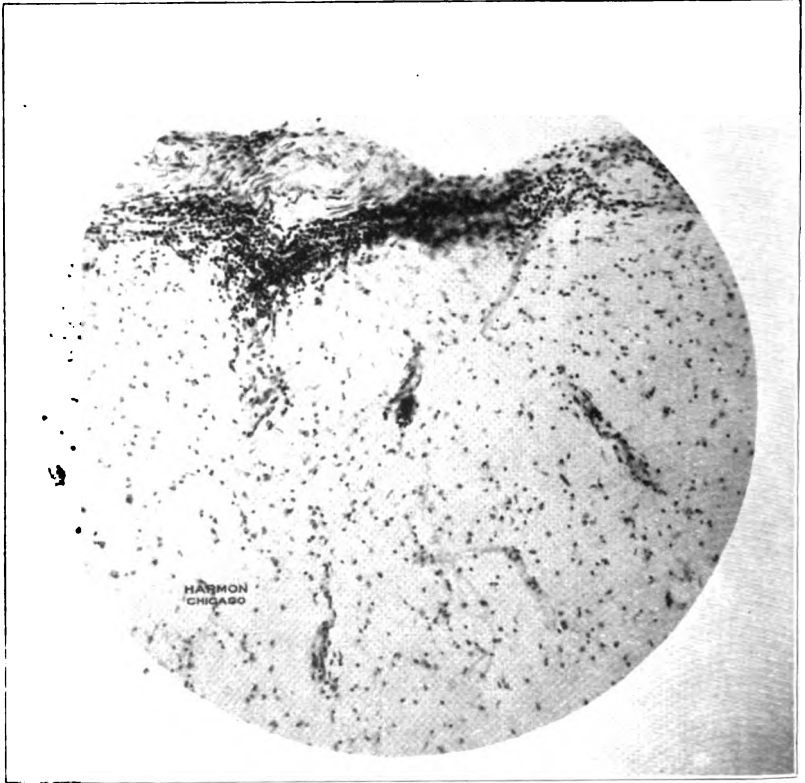


Fig. 3.—Infiltration of pia and vessels of spinal cord. The vascular infiltration is mild. Toluidin blue stain; $\times 110$.

revealed quite an unexpected amount of changes which briefly can be summed up as follows (Fig. 4) :

1. There were numerous nerve fibers with perfectly normal myelin sheath and axon.
2. A number of fibers were deprived of myelin and consisted only of tortuous and slightly swollen axons.
3. Fragments of myelin, so-called Marchi globules, lodged within larger or smaller vacuoles surrounded by glia fibers.

4. Vacuoles filled with gliogenous formations described by Jacob⁴ as myeloclasts and myelophages.

5. There was an abundance of glia fibers covered with glia nuclei and large protoplasmic glia cells with one or two nuclei frequently containing fat droplets and many small processes.

6. There were gitter cells of various types as described by Jacob, some containing a large vacuole with fragments of myelin or axon (gitter cells-*a*);

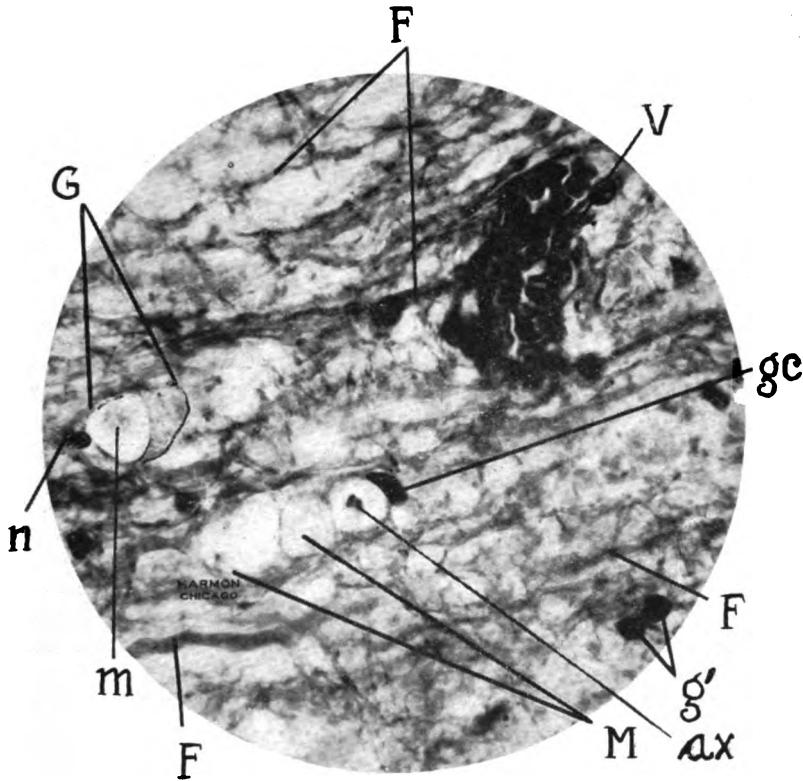


Fig. 4.—Longitudinal section of the lateral pyramid of the spinal cord. Normal fibers (*F*) are separated by a great number of vacuoles, some of which contain remnants of nerve fibers (myelin and axons); *F*, *F*, nerve fibers; *V*, vessel; *G*, gitter cell-*a*; its right half is like a typical gitter cell where with the help of a lens one can see its vacuolated structure, the left half is represented by a vacuole containing a fragment of myelin (*m*); *n*, its nucleus; *gc*, glia cell with a dark nucleus and processes enveloping a fragment of axon (*ax*); *g'*, glia nuclei; *M*, myelin globules (Marchi globules). Alzheimer-Mann stain; $\times 600$.

4. Jacob, A.: Ueber die feinere Histologie der secundären Faser-degeneration in der weissen Substanz des Rückenmarks, Nissl-Alzheimer's Arbeiten 5:182, 1912.

these cells were the most numerous; other glitter cells showed some large processes (glitter cells- β) and very few typical glitter cells round in shape without any processes and densely packed with fat globules (glitter cells- γ).

7. Numerous vacuoles devoid of contents giving the section an areolar sieve-like appearance were found.

Of the outlined changes the so-called Marchi globules were the most frequent, indicating the degenerative process to be in the earliest stages. The same changes, but much less pronounced, could be found in the pyramids of the medulla, while the posterior columns, the spino-cerebellar tracts and various regions of the medulla showed no changes at all, except excessive vascularization and dense glia formation around the vessels.

Especially interesting were changes in the cortical motor area, as well as in the gray matter of the medulla and spinal cord. The latter revealed scarcity and atrophy of the anterior horn ganglion cells, abundance of glia fibers and glia nuclei, some amyloid bodies and excessive vascularization; in short, changes analogous to those in amyotrophic lateral sclerosis. The nuclei of the medulla (the twelfth, tenth and ninth were principally studied) showed a number of cells more or less preserved, but as a rule there was a great lack of ganglion cells. The striking feature was abundance of vessels in the nuclei (Fig. 5) which were literally studded with small new-formed capillaries, some vessels having been congested, but not infiltrated. The same abundance of vessels was seen in the calamus scriptorius, formatio reticularis, nucleus ambiguus and olivary bodies. The elastica stain of Weigert shows especially well the extraordinary number of vessels even as far as the corpora quadrigemina and cerebral cortex. The motor area of the latter, in addition, exhibited marked structural changes in some cells, though many ganglion cells, especially of the upper layers, appeared quite normal. Many cells of the deeper strata (fourth to sixth) were uniform in their staining, granular, broken up, with a pale dislocated nucleus, sometimes invaded by glia cells, etc.; in short, there were neuronophagia, chromatolysis, cell-shrinking, satellitosis which probably accounted for the described condition in the pyramidal tracts.

COMPARISON OF AUTHOR'S CASE WITH BULBAR PARALYSIS AND AMYOTROPHIC LATERAL SCLEROSIS

This combination of changes in pyramidal tracts and anterior horn cells certainly justifies a diagnosis of amyotrophic lateral sclerosis. Yet, when compared with changes as described by me⁵ in a typical case of amyotrophic lateral sclerosis, there could be pointed out some differences. Thus, in amyotrophic lateral sclerosis the pyramidal fibers in the lateral, and partly in the anterior, columns were densely packed with fat-like (lipoid) globules, in Herxheimer (scarlet red) and Marchi specimens (Fig. 6), while in the present case such changes were few and slight. The degeneration evidently did not progress far enough to furnish products that would stain with osmic acid or scarlet

5. Hassin, G. B.: Histopathological Changes in a Case of Amyotrophic Lateral Sclerosis, *Med. Rec.* 91:228 (Feb. 10) 1917.

red, but only such small fragments as could be demonstrated with Alzheimer-Mann, or Mallory-Jacob stains. In typical amyotrophic lateral sclerosis the glia changes are much more striking, resembling those found in various stages of experimental secondary degeneration so well described by Jacob. On the other hand, vascular changes, especially in the affected nuclei, are missing or at least not recorded in amyotrophic lateral sclerosis. Their presence in bulbar paralysis has been pointed out by Duchenne and Joffroy,⁶ and Eisenlohr.⁷ In amy-

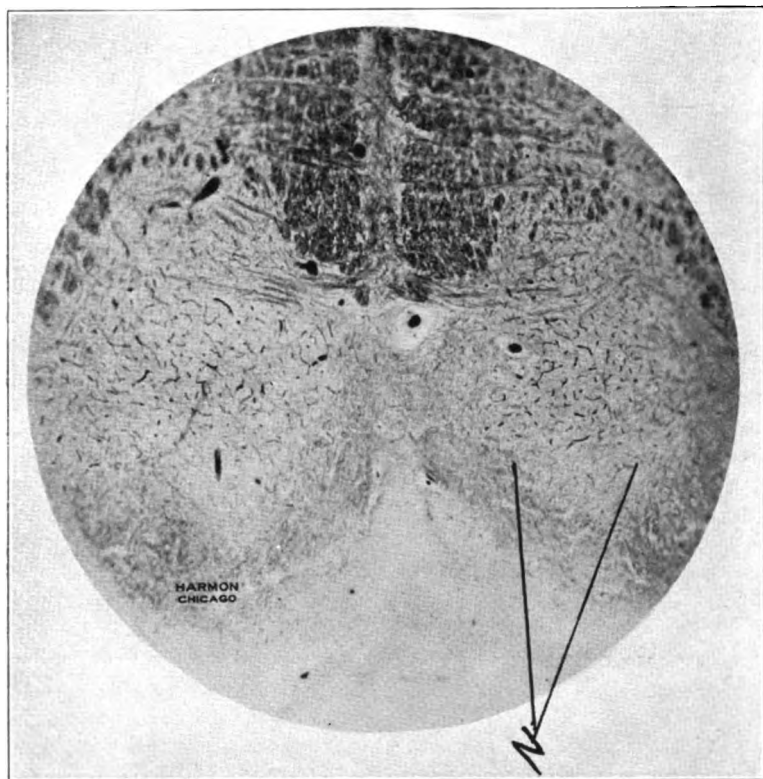


Fig. 5.—The nuclei of the twelfth nerve, separated in the front by the medial fillet. The anterior three fourths of the nuclei exhibit an abundance of vessels; the posterior fourth (*N*) is quite normal, appearing pale and arched. Weigert-Pal stain; $\times 27$.

otrophic lateral sclerosis many vessels show infiltration of their adventitial spaces with fat which I could not find in this case, in which,

6. Duchenne and Joffroy: De l'atrophie aiguë et chronique des cellules nerveuses de la moelle et du bulbe rachidien, *Arch. de physiol.* **3**:497, 1870.

7. Eisenlohr: Klinische und Anatomische Beiträge zur progressiven Bulbärparalyse, *Ztschr. f. klin. Med.* **1**:435, 1880.

however, the vessels, especially of the spinal cord and cortex were markedly infiltrated with lymphocytes. The pia in the present case also appeared infiltrated, exclusively with lymphocytes, which is not the case in amyotrophic lateral sclerosis. The foregoing differences, however, are but slight, probably accidental (like, for instance, the pial infiltration), being quantitative rather than qualitative, and a diagnosis of amyotrophic lateral sclerosis in the present case safely could be made. The outlined pathologic changes thus afford additional proof

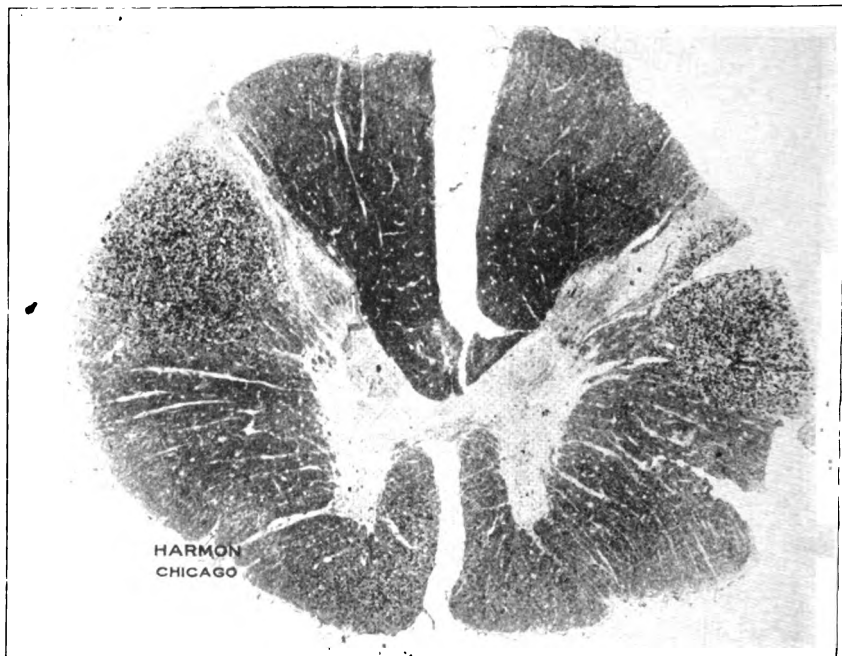


Fig. 6.—Tenth dorsal segment from a patient with amyotrophic lateral sclerosis. The lateral and somewhat the anterior pyramids are converted into a mass of drops and droplets of fat. No fat globules are to be found in the posterior columns, Gowers' or Flechsig's tracts, or in the lateral limiting layer of either side. A frozen section photographed four years after it was stained with Herxheimer scarlet red stain. A hand lens will bring out the details better; $\times 15$.

that bulbar paralysis is practically nothing but amyotrophic lateral sclerosis. Carefully studied cases of chronic bulbar paralysis without concomitant pyramidal tract involvement are practically unknown. The few cases reported (Charcot,⁸ Duchenne and Joffroy,⁶ Leyden,⁹

8. Charcot, J.: Note sur un cas de paralysie glosso-laryngée, *Arch. de physiol. norm. et path.* **3**:247, 1870.

9. Leyden, E.: Progressive Bulbärparalyse, *Arch. f. Psychiat.* **2**:429, 643, 1870.

Eisenlohr⁷) in which the lateral columns were found intact are the earliest recorded, and it is probable, as Leyden himself admits, that possibly in these cases the pyramidal changes were overlooked. Amyotrophic lateral sclerosis may begin and run its course as typical bulbar paralysis (Hun,¹⁰ Maier,¹¹ Kussmaul,¹² Leyden,¹³ Dejerine¹⁴), though a majority of cases recorded show that it *follows* the lateral column lesion. In our case, bulbar and pyramidal lesions apparently occurred simultaneously. The simultaneous combined lesion of the motor area of the brain, of the motor nuclei of the bulb and spinal cord will give a clinical picture of amyotrophic lateral sclerosis.⁵ But it may happen that the medullary nuclei will be *principally*, though *not exclusively*, involved. Then Duchenne's picture of glosso-labio-laryngeal paralysis will obtain. Preponderating involvement of anterior horn cells of the spinal cord will result in progressive muscular atrophy, or subacute general anterior spinal paralysis of Duchenne, also known as chronic anterior poliomyelitis. Finally, it may happen that the cortical motor cells will be principally affected resulting in a peculiar syndrome described as progressive hemiplegia, triplegia, quadriplegia, etc. Whatever the variety, probably in each case there will be found motor cell changes in the cortex, in the medulla oblongata, pons and spinal cord. The present case demonstrates this combination in bulbar paralysis, it being also present in amyotrophic lateral sclerosis, and Alzheimer¹⁵ showed the same thing in progressive muscular atrophy. Whether the same applies in progressive hemiplegia remains to be seen, as there are no exhaustive histopathological records of this syndrome.¹⁶ Here it must be pointed out that our knowledge of the histopathology of bulbar paralysis, amyotrophic lateral sclerosis, progressive muscular atrophy and progressive hemiplegia is yet in a very

10. Hun, E. R.: Labio-glosso-laryngeal Paralysis, *Am. J. Insan.* **28**:194, 1871.

11. Maier, R.: Ein Fall von vortschreitender Bulbärparalyse, *Virchow's Arch. f. Path. Anat.* **8**:641, 1878.

12. Kussmaul, S.: Ueber fortschreitende Bulbärparalyse und ihre Verhältnisse zur progressiven Muskelatrophie, *Samml. klin. Vortr. (Volkmann's)* **1**:439, No. 54, *Innere Medizin*.

13. Leyden, E.: Ueber progressive amyotrophische Bulbärparalyse und ihre Beziehungen zur symmetrischen Seitenstrangsklerose, *Arch. f. Psychiat.* **8**:641, 1878.

14. Dejerine, J.: Etude anatomique et clinique sur la paralysie labio-glosso-larynge, *Arch. de physiol. norm. et path.* **152**:180, 1883.

15. Alzheimer, A.: Ueber einen Fall von spinaler progressiver Muskelatrophie mit hinzutretender Erkrankung bulbärer Kerne und der Rinde, *Arch. f. Psychiat.* **23**:459, 1892.

16. Hassin, G. B.: A Case of Amyotrophic Lateral Sclerosis in the Form of Progressively Developing Triplegia, *New York M. J.* **106**:932 (Nov. 17) 1917.

unsatisfactory state, contributions to these problems are not only very sparse, but very incomplete. Thus, in the majority of them, there is no record of the changes in the brain or other portions of the central nervous system, which, if studied at all, were followed up with the old methods, while the condition of the glia was not fully described. Further studies with the newer methods are absolutely essential for establishing the real relationship of these four syndromes which clinically differ, in my opinion, by the localization of the parts *principally*, but not *exclusively* involved.

31 North State Street.

LETHARGIC ENCEPHALITIS *

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For the past nine years we have had at the research laboratory of the health department of New York City a meningitis division, the function of the members of which is to see in consultation all kinds of meningeal conditions for differential diagnosis and treatment. In this connection we have seen over 1,000 cases of meningitis of various kinds, 600 or more cases of poliomyelitis and over 700 cases of meningism with various diseases, besides small numbers of cases of numerous other conditions, so that we have had a fairly good background for the study of a new type of meningeal or cerebral disease.

Last October, as a member of the research laboratory of the health department of New York City, I began to see a new type of disease. The majority of these patients gave a history of influenza followed in a varying length of time by headache, drowsiness and apathy, usually accompanied by a low irregular fever, strongly suggesting a slowly developing tuberculous meningitis. One of the earliest patients, "X," showed extreme restlessness instead of drowsiness, marked muscular weakness and some paralysis of the cranial nerves. These conditions suggested a variety of diagnoses. Meningism seemed possible in certain instances, perhaps caused by some gastro-intestinal disorder, or it may have followed, instead of accompanied, influenza; this, however, was ruled out as the spinal fluid showed a marked increase in the protein elements and cells instead of being normal, as is the rule in meningism. Syphilitic disease was suspected, especially in "X," but this was disproved by the negative Wassermann test and by the character of the gold chlorid curve. Brain tumor must be differentiated in certain of the more severe cases, and this differentiation has proved a stumbling block to some very eminent neurologists. Tuberculous meningitis was considered in many cases, but this diagnosis was discarded because of failure to find the tubercle bacillus either by smear or by animal inoculation, the normal reduction of Fehling's solution and the favorable termination. In some instances the encephalitic type of poliomyelitis was suggested by the clinical picture and by the spinal fluid findings, but this type is rare even in epidemics of poliomyelitis,

* Read before the Section on Preventive Medicine and Public Health at the Seventieth Annual Session of the American Medical Association, Atlantic City, N. J., June 11, 1919.

* From the Research Laboratory Department of Health, New York City.

TABLE 1.—SALIENT FEATURES OF FORTY CASES OF LETHARGIC ENCEPHALITIS

Case No.	Age	History of Influenza	Type of Onset	Lethargy	Asthenia	Headache	Falises	Reflexes	Temperature	Vomiting	Miscellaneous	Duration	Outcome	Remarks
14	12 wk. M.	+	Slow	Slight	—	—	100	+	?	Recovered	
38	8 mo. M.	+	Slow	Int. strabis.	—	102	—	7 days	Died	
38	9 mo. F.	—	Sudden	—	To 105	—	Mask-like expression, spas-	2 weeks	Recovered	
32	15 mo. M.	+	Slow	+	+	—	Slight	+	tically, marked tremors, convulsions just before death, marked arrhythmia	3 weeks	Died	
5	15 mo. M.	+	Slow	+	+	—	?	+	+	Tremors.....	4-5 weeks	Recovered	
9	18 mo. F.	+	Slow	+	Int. strabis.	N.	102-103	—	1 week	Died	
371	20 mo. F.	+	Slow	+	Int. strabis. rt. facial	—	Slight	+	3 months	Died	
94	2 yrs. M.	+	Slow	+	+	—	N.	101	+	Rotating of eyeballs, twist-	10 days	Recovered	
49	2 yrs. M.	+	Slow	+	?	—	104	+	ing of lips, difficult respi-	6 days	Died	
											ration, convulsions, ar-			
											rhythmia			
54	3 yrs. M.	+	Sudden	+	+	+	—	104	+	4 weeks	Recovered	
16	3 yrs. M.	+	Slow	+	+	—	Slight	+	3 weeks	Recovered	
87	3½ yrs. M.	—	Sudden	+	+	—	100	+	10 days	Recovered	
351	4 yrs. F.	Prob-	Sudden	+	?	—	Slight	+	Less than 1 month	Recovered	
		able											
137	4 yrs. M.	—	Sudden	+	?	—	Slight	+	2 weeks	Recovered	
391	6½ yrs. F.	+	Slow	+	+	N.	100.5	—	2 weeks	Recovered	
385	7 yrs. F.	+	Slow	+	+	+	101.5	—	2-3 weeks	Recovered	
19	7 yrs. M.	+	?	+	+	—	104	—	Tremors.....	3 weeks	Died	
41	7 yrs. M.	+	Slow	+	+	— left	104	—	Spasticity of legs, vacant expression	4 weeks	Recovered	
								N. rt.					
80	9 yrs. M.	—	Sudden	+	+	+	N.	Slight	—	Babinski.....	2 weeks	Recovering	
357	9 yrs. M.	+	Slow	+	+	+	104	—	1 month	Recovered	
46	9 yrs. M.	—	Slow	+++	+++	Facial Slight dysphagia	+	+	—	Babinski, mask-like expres-	8 months	Improving	
											sion, tremors at times, arrhythmia			
35	10 yrs. M.	+	Sudden	+	+	+	—	+	+	2 weeks	Recovering	
100	10 yrs. M.	+	Slow	+	+	+	N.	Slight	+	7-8 days	Died	
38	11 yrs. M.	+	Slow	+	+	+	Rt. facial	D.	101	—	Babinski, pupils unequal, Delirious during convales-	2 months	Recovered	Mental examination showed it had been abnormal some years, not sequel of encephalitis

331	14 yrs. M.	?	Sudden	+	+	—	102	+	Delirium.....	3 weeks	Recovered
382	16 yrs. M.	—	Sudden	+	+	—	To 106.6	—	3 days	Died
Y	18 yrs. M.	—	Sudden	++	++	—	102	—	Slight delirium at times.....	3-4 weeks	Recovered
24	20 yrs. M.	+	Slow	+	+	Rt. facial	—	103	—	Paralysis after 1 week, delirium	1 month	Recovered
48	20 yrs. M.	—	Sudden	+++	+++	Dysphagia	—	103	..	Leg spastic, marked sweating, mask-like expression, tremors, delirium	1 week	Died
353	23 yrs. M.	+	Slow	++	++	++	+	+	1 month	Recovered
383	25 yrs. F.	+	Slow	+++	+++	Facial	103	+	Pregnant, normal delivery, delirium at times	Over 2 months	Recovered
66	30 yrs. M.	+	Slow	+++	+++	Double facial	++	90-103	—	Restlessness, marked tremors, mask-like expression	6 weeks	Recovered
103	30 yrs. M.	+	Slow	+	+	Left facial	N.	—	—	Disturbance of vision, vacant expression, delirium at times	?	Recovering
27	32 yrs. M.	+	Slow	+++	+++	Bilat. facial	D.	102	—	Nystagmus, dizziness, diplopia, tremors, mask-like expression, spasticity	4 months	Recovered
50	35 yrs. M.	—	Slow	++	++	Left facial Dif. speech, arm and leg paralyzed	++	101-102	—	Diplopia, difficult speech, dizziness	2 months	Recovering
72	43 yrs. M.	+	Sudden	++	++	Rt. arm	N. left D. rt.	—	—	Dizziness, diminished sensibility on right upper and lower extremities	2 weeks	Recovering
382	45 yrs. M.	+	Slow	++	++	Ptosis of rt. eyelid	++	103	—	B a b l e s k i Incontinence, sweating, mask-like expression, delirium	4 months	Recovered
68	50 yrs. M.	—	Slow	+++	+++	Sl. ocular	D.	Slight	—	Babinski, mask-like expression	3 months	Recovered
X	50 yrs. M.	+	Slow	—	+	Facial Dif. speech	—	+	+	Irritability, pain, restlessness	3 mo.; facial par. after 5 mo., nearly gone at 6	Slight paralysis remaining recovered
52	50 yrs. M.	—	Sudden	++	++	N.	Slight	—	1 month	Died

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Case No.	Age	History of Influenza	Type of Onset	Lethargy	Asthenia	Headache	Falsies	Reflexes	Temperature	Vomiting	Miscellaneous	Duration	Outcome	Remarks
14	12 wk. M.	+	Slow	Slight	—	—	100	+	Slight convulsions.....	?	Recovered	
38	8 mo. M.	+	Slow	Int. strab.	—	102	—	Irritability.....	7 days	Died	
38	9 mo. F.	—	Sudden	—	TO 105	—	Mask-like expression, spas-	2 weeks	Recovered	
32	15 mo. M.	+	Slow	+	+	—	Slight	+	tletty, marked tremors, convulsions just before death, marked arrhythmia	3 weeks	Died	
5	15 mo. M.	+	Slow	+	+	—	?	+	+	Tremors.....	4-5 weeks	Recovered	
9	18 mo. F.	+	Slow	+	Int. strab.	N.	102-103	—	Convulsions.....	1 week	Died	
371	20 mo. F.	+	Slow	rt. facial	—	Slight	+	3 months	Died	
94	2 yrs. M.	+	Slow	+	+	—	N.	101	+	Rotating of eyeballs, twist-	10 days	Recovered	
49	2 yrs. M.	+	Slow	+	?	—	104	+	ing of lips, difficult respi-	6 days	Died	
											ration, convulsions, ar-			
											rhythmia			
54	3 yrs. M.	+	Sudden	+	+	+	—	—	104	—	Convulsions.....	4 weeks	Recovered	
16	3 yrs. M.	+	Slow	+	+	—	Slight	+	3 weeks	Recovered	
87	3½ yrs. M.	—	Sudden	+	+	—	100	+	10 days	Recovered	
351	4 yrs. F.	Prob-	Sudden	+	?	+	—	Slight	+	Convulsions.....	Less than 1 month	Recovered	
137	4 yrs. M.	able	Sudden	+	?	+	—	Slight	+	Convulsions.....	2 weeks	Recovered	
301	6½ yrs. F.	+	Slow	+	+	N.	104.5	—	Tremors.....	2 weeks	Recovered	
305	7 yrs. F.	+	Slow	+	+	—	101.5	—	2-3 weeks	Recovered	
19	7 yrs. M.	+	?	+	+	—	104	—	Spasticity of legs, vacant expression	3 weeks	Died	
41	7 yrs. M.	+	Slow	+	+	N. left	104	—	Babinski.....	4 weeks	Recovered	
80	9 yrs. M.	—	Sudden	+	+	+	N.	Slight	+	Babinski.....	2 weeks	Recovering	
357	9 yrs. M.	—	Slow	+	+	+	104	+	Babinski, mask-like expres-	1 month	Recovered	
46	9 yrs. M.	—	Slow	+	+	Facial Slight dysphagia	+	+	+	sion, tremors at times, arrhythmia	3 months	Improving	
35	10 yrs. M.	+	Sudden	+	+	+	N.	+	+	Convulsions.....	2 weeks	Recovering	
100	10 yrs. M.	+	Slow	+	+	+	D.	Slight	+	Babinski, pupils unequal....	7-8 days	Died	Mental examination showed it had been abnormal some years, not sequel of en-
38	11 yrs. M.	+	Slow	+	+	+	Rt. facial	—	101	—	Delirious during convales-	2 months	Recovered	ence, arrhythmia at times

331	14 yrs. M.	?	Sudden	+	+	+	—	102	+	Delirium.....	3 weeks	Recovered
332	16 yrs. M.	—	Sudden	+	+	+	—	To 106.6	—	3 days	Died
Y	18 yrs. M.	—	Sudden	++	++	+	—	102	—	Slight delirium at times.....	3-4 weeks	Recovered
24	20 yrs. M.	+	Slow	+	+	+	Rt. facial	—	103	—	Paralysis after 1 week, delirium	1 month	Recovered
48	20 yrs. M.	—	Sudden	+++	+++	+	Dysphagia	—	103	..	Leg spastic, marked sweating, mask-like expression, tremors, delirium	1 week	Died
353	23 yrs. M.	+	Slow	++	++	+	++	+	+	1 month	Recovered
383	25 yrs. F.	+	Slow	+++	+++	+	Facial	103	+	Pregnant, normal delivery, delirium at times	Over 2 months	Recovered
66	30 yrs. M.	+	Slow	+++	+++	++	Double facial	++	90-103	—	Restlessness, marked tremors, mask-like expression	6 weeks	Recovered
103	30 yrs. M.	+	Slow	+	+	+	Left facial	N.	—	—	Disturbance of vision, vacant expression, delirium at times	?	Recovering
27	32 yrs. M.	+	Slow	+++	+++	+	Bilat. facial	D.	102	—	Nystagmus, dizziness, diplopia, tremors, mask-like expression, spasticity	4 months	Recovered
50	35 yrs. M.	—	Slow	++	++	+	Left facial Dif. speech, arm and leg paralyzed	++	101-102	—	Diplopia, difficult speech, dizziness	2 months	Recovering
72	43 yrs. M.	+	Sudden	++	++	+	Rt. arm	N. left D. rt.	—	—	Dizziness, diminished sensitiveness on right upper and lower extremities	2 weeks	Recovering
392	45 yrs. M.	+	Slow	++	++	+	Prosis of rt. eyelid	++	103	—	B a b i n s k i Incontinence, sweating, mask-like expression, delirium	4 months	Recovered
68	50 yrs. M.	—	Slow	+++	+++	—	Sl. ocular	D.	Slight	—	Babinski, mask-like expression	3 months	Recovered
X	50 yrs. M.	+	Slow	—	+	+	Facial Dif. speech	—	+	+	Irritability, pain, restlessness	3 mo.; facial par. after 5 mo. nearly gone at 6	Practically recovered
52	50 yrs. M.	—	Sudden	++	++	+	N.	Slight	—	1 month	Died

Mental depression, slight paralysis

Slight weakness

Rapid pulse

Slight paralysis remaining

and during the fall and winter the number of cases of even the spinal type has been extremely small. Finally, I began calling these cases influenzal encephalitis, since the symptoms were those of encephalitis and since they so often followed influenza. Not until midwinter did I designate these cases lethargic encephalitis, as it was not until that time that I encountered patients with lethargy, asthenia and oculomotor palsies, the characteristic triad of symptoms described by the English and French. Table 1 gives some of the salient features of forty cases in which the diagnosis seems to be well established. A study of the table shows: (1) wide age distribution—from twelve weeks to over fifty years. From the nature of our work we see an unusually high proportion of children. (2) The large proportion of males, thirty-three out of forty. Tucker reports nine males out of eleven cases. In other reports, usually of smaller numbers, there has not been this marked difference. (3) The history of an attack, clinically influenza, in twenty-seven of the forty cases. It will be noted that the onset is more often slow than sudden, and that lethargy and asthenia are nearly constant symptoms, while cranial nerve or other palsies are present in less than half of the cases. It is possible that our list includes a rather high proportion of mild cases, since we are called in consultation where an early tuberculous meningitis is suspected. Where lumbar puncture is not so freely resorted to, I fancy that many of these milder cases are unrecognized.

REPORT OF CASES

Certain patients are perhaps of sufficient interest to deserve special attention.

CASE 1.—Patient 357 had a typical mild case. He had influenza early in October. About the middle of October he became gradually worse, with headache, vomiting, constipation (which the French call the meningitic triad), irregular fever (102 to 104 F.), and marked apathy. This history was very suggestive of tuberculous meningitis. I saw him October 27. He was then somewhat improved. His mental condition was nearly normal, there was no well-marked stiffness of the back, but he still had fever, his patellar reflexes could not be obtained, and there was a moderate Kernig's and Macewen's sign. Lumbar puncture revealed clear fluid under pressure showing only a slight increase in the proteid contents. The case cleared up quite promptly.

CASE 2.—Patient 66 was a physician about 30 years of age. He had an attack of influenza which began February 18 and lasted for six or seven days. While there was general improvement after the attack, he continued to suffer with severe headache, and late in February he began to have fever, which ran as high as 103 F., and marked apathy. I saw him March 12. His face was entirely expressionless and he made no response whatever when requested to smile; apparently there was great weakness of the facial muscles on both sides. At times there was ptosis. He was unable to move, but frequently asked to be turned as he was uncomfortable if left long in one position as a tremor involving all the limbs was then most likely to develop. His pupils

were equal and reacted to light; the knee jerks were slightly increased and he was entirely clear mentally and very anxious about his condition. As Dr. Foster Kennedy very aptly expresses it, these patients are emotionally stuporous and intellectually bright. About 35 c.c. of clear spinal fluid was withdrawn, showing great increase in cells and proteid contents; a negative Wassermann test was made. He was able to be out of bed by the end of March and after another month had quite recovered, though he was still a little weak and suffered slightly from insomnia.

CASE 3.—Patient 393, five months pregnant, had an attack of influenza two weeks before the onset of the encephalitis, which began gradually early in December with headache, chill and fever, vomiting, sweating and delirium. I saw her December 14, at which time she was stuporous. There was some stiffness of the neck, and a right facial paralysis. About 25 c.c. of clear spinal fluid were withdrawn, which showed great increase in cells and in the protein elements, and a negative Wassermann test. A guinea-pig inoculated with the fluid gave a negative reaction for tuberculosis. Her condition remained the same for two weeks or more and then she gradually recovered. The facial paralysis cleared up, and she had a normal delivery at term.

CASE 4.—Patient 38, a boy of 11 years, was seen February 4. Late in January he had what had been diagnosed as a mild attack of influenza. On the 31st he began to have gradually increasing headache, apathy, and low fever. When examined, February 4, he was stuporous, the pulse rapid (120), and quite arrhythmic. His temperature was between 100 and 101 F. The right pupil reacted sluggishly to light, the left was normal. The right patellar reflex was increased, and there was a right facial paralysis. Fifteen c.c. of spinal fluid withdrawn on this occasion and 25 c.c. withdrawn two days later showed slight increase in cells, albumin and globulin. The Wassermann test was negative. The patellar reflexes became equal in a few days. By the 11th he was greatly improved. The stuporous condition had disappeared, and the pulse, though still rapid, had ceased to be arrhythmic. Later, however, he became mentally disturbed and was violent. By the middle of April, he seemed to have recovered, but his mother reported that he had a voracious appetite and was very troublesome and difficult to manage. At first it seemed that his condition was due to the encephalitis but an investigation of the case revealed the fact that he had been in classes for atypical children for two or three years. He was examined by Dr. Coffin of the department of education for New York City, and was found to belong to the hyperactive, precocious type of children that it is so difficult to deal with satisfactorily.

CASE 5.—Patient 46, a boy of 9 years, is of interest on account of the long duration of his disease. He was admitted to Willard Parker Hospital with a history of being ill for three days. He was then comatose, had slight rigidity of the neck and Kernig's sign, exaggerated knee jerks, positive Brudzinski sign and Babinski reflex. He ran an irregular temperature, from 100 to 103 F. until February 19, after which it was below 100 until March 14. At times his pulse and respiration were irregular. He became progressively worse and by February 20 had marked rigidity of the entire body, and a mask-like, expressionless face. He became unable or unwilling to swallow and had to be tube fed. The white blood count was 15,000, 81 per cent. polymorphonuclears. The first lumbar puncture, February 13, showed a clear fluid with moderate increase in cells, 60 per cent. polymorphonuclears, moderate increase in protein elements, normal reduction in Fehling's sign and negative Wassermann test. Fluid withdrawn February 14, was slightly blood-tinged so that it had a

somewhat hazy appearance. Therefore, with the clinical picture resembling meningitis so strongly, and an excess of polymorphonuclears in the first fluid, serum was given. Of course, this obscured the spinal fluid picture for some time. During all this time the child did not speak and has not up to the present time. There were frequent muscular twitchings. Early in March he began to move his head and a little later his legs and arms, and seemed to be progressing toward recovery. On March 14, however, the temperature rose to 106 F., he perspired profusely and seemed to be in a desperate condition. A blood culture at this time was negative. Twenty c.c. of spinal fluid were withdrawn under some pressure, showing a moderate increase in cells, 80 per cent. mononuclears, increase in albumin and globulin, and a normal reduction of Fehling's. On March 15 he began to improve; his temperature dropped to 100 F. Since that time his condition has shown some improvement, and he has gained in weight. A peculiar hairiness has appeared on the trunk, legs, arms and forehead. He looks about and has an intelligent expression but does not respond in any way when spoken to. He still has to be tube fed but can swallow if one is dexterous enough to insert food when his mouth is open. His arms, and especially his legs, are still somewhat spastic and the right leg shows contracture, though it is now possible nearly to straighten it without his evidencing much discomfort. Being naturally optimistic, we are expecting his ultimate recovery.

CASE 6.—The patient (48) 20 years old, became ill while he was in a hospital being treated for flatfoot. The onset, which was sudden, came on February 13, with severe pain in the chest, side, and shoulder, temperature of 102 F., pulse from 80 to 100. February 14 the condition was about the same except that he complained of pain in his eyes and during the night he became irrational. On the 15th and 16th the condition became worse, with severe headache and active delirium. His jaw was so rigid that his throat could not be examined and the arms and legs became spastic. There was slight rigidity of the neck and the Kernig sign was present, which may have been due to the general spasticity. The patellar reflexes were present, the pupils reacted rather sluggishly to light, there were a few large, moist râles in the right base posteriorly and no bronchial breathing. The pulse was rapid and of poor quality at times. His temperature went as high as 104 F. A diagnosis of cerebrospinal meningitis was made and an unsuccessful attempt was made to perform a lumbar puncture. The patient was sent to the Willard Parker Hospital on the evening of February 16. Twenty c.c. of clear fluid were withdrawn showing a moderate increase in cells and mononuclears, albumin and globulin greatly increased, and a normal Fehling's reduction. There were no organisms by smear or culture and a negative Wassermann reaction. The second puncture, February 19, showed practically the same picture except that there was a greater increase in cells. The blood count was normal, 10,000 leukocytes, 65 per cent. polymorphonuclears. His temperature ranged from 103 to 105 F. His pulse was between 120 and 130, but was not irregular until slightly before his death, which occurred February 20. He lay on his back with his eyes tightly closed and his whole body markedly spastic. His neck was rigid. It was impossible to obtain the knee jerks, but the plantar reflexes were increased. At times there was a tremor, especially of the legs. He was generally comatose but at intervals there was muttering delirium, and he frequently perspired freely. He was able to swallow until near death though he often refused unless urged to do so by one of the nurses whose directions he usually followed. His death was described as being particularly

painful with evidences of respiratory difficulty, probably of central origin, since it did not develop until shortly before he died. There was no evidence of paralysis at any time. A necropsy was performed which showed marked congestion of all the organs. The report on the brain and upper part of the cord will be given under pathology.

CLINICAL PICTURE

The characteristic clinical picture of lethargic encephalitis is generally as follows: a gradual onset, often following influenza, a low irregular fever, headache, marked lethargy and asthenia, with or without cranial nerve palsies.

TREATMENT

While there is no specific treatment, a lumbar puncture has been followed in many cases by temporary improvement. The spinal fluid is usually under increased pressure and it seems to me desirable to relieve it. Of course, every effort should be made to keep the patient comfortable and general eliminative and supportive measures should be carried out. Symptomatic treatment should be instituted as the indications arise.

LABORATORY FINDINGS

The blood picture is not characteristic. It is usually normal or shows a slight leukocytosis, perhaps up to 15,000. Blood cultures are sterile. The urine is usually negative (Table 2). The spinal fluid shows practically the same picture as in poliomyelitis. The cells are usually slightly or moderately increased, seldom greatly, perhaps up to 150 to 200 in some cases. While cell counts may run higher in poliomyelitis, the great majority do not. As in poliomyelitis, there is usually an excess of mononuclears, but an excess of polymorphonuclears may occur. The albumin and globulin are greatly increased, the reduction in Fehling's is normal. The increase in cells and protein content is not always in the same ratio. No organisms are shown by smear or culture. The gold chlorid curve depends on the amount of albumin and globulin present and duplicate curves may be selected from those in poliomyelitis fluids. In some instances, most often in convalescent or mild cases, the findings may depart little from the normal. This is true also in poliomyelitis. Most reports of encephalitis show that the cell count (which, unfortunately, is often the only information given) falls off very quickly. In two of our cases of long duration, the character of the fluid did not change materially over a period of several weeks, but the condition of the patients also showed little change. This comparison with poliomyelitis is made, not because I believe the two diseases are at all identical, but to emphasize the fact that in each instance the spinal fluid is not specific, but shows the

reaction of the meninges to an inflammation of the brain substance. A somewhat similar condition exists in the various syphilitic involvements of the central nervous system, but in these conditions the gold chlorid curve and the Wassermann test are helpful in making the diagnosis.

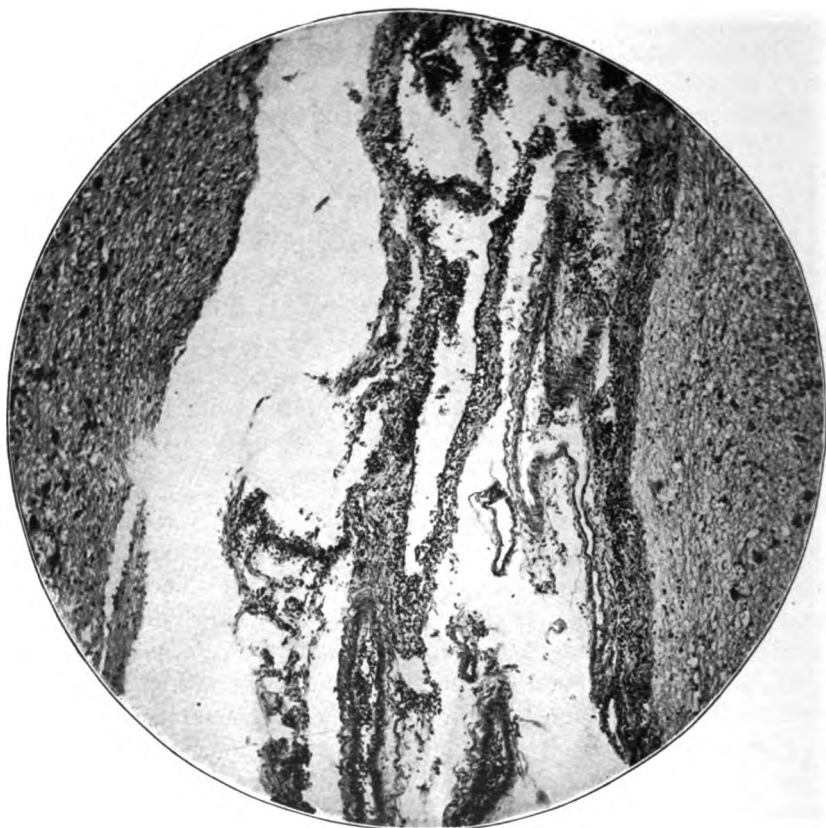


Fig. 1.—Meninges of the anterior fissure of the cord, showing fibrosis and round-cell infiltration, mostly in the vessel walls. Diffuse round-celled infiltrations of the arcuate nucleus of the pyramids is shown in either side.

The most difficult and the most needed diagnosis is that made from the fluid of tuberculous meningitis. While generally the number of cells and the increase in albumin and globulin is greater in the latter disease, it is by no means always so, and it is sometimes necessary to examine more than one fluid before one can be certain of the diagnosis, as it is often difficult to find the tuberculosis bacillus in early tuberculous meningitis and the reduction of Fehling's may be normal at that time. The following table shows the findings in the spinal fluids in our cases of lethargic encephalitis.

TABLE 2.—LABORATORY FINDINGS IN CASES OF LETHARGIC ENCEPHALITIS

Case No.	Amount in C.e.	Cytology	Protein	Fehling's Reduction	Animal In-oculation	Wassermann Reaction	Onset to Puncture	Gold Chlorid
14	30	Greatly increased..... Mononuclears 80%	+++	+++	?	
388	25	Greatly increased..... Mononuclears 90%	++++	—	8 days	
368	15 sl. cloudy	Greatly increased..... Mononuclears 90%	++	+++	8 days	
32	60	Greatly increased..... Mononuclears 80%	++ 1	+++	—	—	14 days	1234321000
	35	Greatly increased..... Mononuclears 80%	+++	+++	15 days	
	30	Slight to moderate increase, Monos. 90%	+++	++	16 days	
	30	Greatly increased..... Mononuclears 80%	++++	++	18 days	
5								
9	30	No increase.....	+ 1	+++	5 days	
371	10	Greatly increased..... Mononuclears 80%	++	+++	18 days	
94								
49	12	Slight increase.....	+	+++	4 days	
54	5	Slight increase.....	+ 1	+++	5 days	
16	30	No increase.....	+	+++	?	
87	20	Very great increase..... Mononuclears 90%	+	+++	—	4 days	
351	20	No increase.....	++ 1	+++	4 days	
137	10	No increase.....	+ 1	+++	3 days	
391	25	Slight to moderate..... Mononuclears 90%	+ 1	+++	—	—	7 days	
395	15	Slight increase.....	+	+++	4 days	
19	35	Moderate increase..... Mononuclears 90%	++	+++	—	4 days	
41	20	Greatly increased..... Mononuclears 95%	++ 1	+++	3 days	
80								
357	35	No increase.....	+	+++	14 days	
46	15	Moderate increase..... Polys. 60%	++	+++	—	3 days	
	25	Bloody fluid.....	++	+++	4 days	
	30	Bloody fluid.....	++ 1	+++	5 days	
	20	Moderate increase..... Mononuclears 80%	+++	+++	6 days	
	20	Moderate increase..... Mononuclears 80%	++	+++	8 days	
	15	Moderate increase..... Mononuclears 95%	++	+++	32 days	
	20	Slight to moderate.....	+++	+++	50 days	
35	10	Moderate increase.....	+++	+++	—	2 days	
100	20	Very great increase..... Mononuclears 90%	++++	++	—	—	2 days	
	30	Great increase.....	++++	+	4 days	
38	15	Slight increase.....	+	+++	—	4 days	
	25	Slight increase.....	+	+++	6 days	
331	30	Slight increase.....	+	+++	21 days	
382	20	No increase.....	+	+++	—	1 day	
Y								
24	25	Greatly increased..... Mononuclears 80%	++ 1	+++	14 days	
48	20	Moderate increase..... Mononuclears 60%	+++	+++	—	?	
	20	Greatly increased..... Mononuclears 80%	+++	+++		
353	5	Slight increase.....	++	+++	8 days	
393	25	Moderate increase..... Mononuclears 85%	++ 1	++ 1	—	—	10 days	
66	30	Moderate to great incr.	++ 1	+++	—	15 days	
103								
27	30	Moderate increase..... Mononuclears 80%	+ 1	+++	—	2 days	1232100000
59	25	Moderate to great incr.	++ 1	+++	—	14 days	
72	25	No increase.....	+ 1	+++	3 days	
392	20	Greatly increased..... Mononuclears 90%	++	+++	—	—	10 days	
	25	Greatly increased..... Mononuclears 80%	++	++ 1	18 days	
	35	Moderate increase..... Mononuclears 80%	++	+++	20 days	
	20	Moderate to great incr. Mononuclears 90%	++	+++	44 days	
68								
X	30	Slight increase.....	+++	+++	—	7 days	00012455560
52	30	Moderate increase.....	+	+++	—	17 days	

The inoculation of monkeys with the emulsified brain and cord of fatal cases has given far from uniform and conclusive results. The English reported failures in their attempts, though the same workers had been almost uniformly successful in dealing with material from cases of poliomyelitis. Von Weisner reported an instance in which the infected monkey died in forty-six hours, his brain on necropsy showing gram-positive cocci. The short period of incubation and the



Fig. 2.—Meninges of the cortex showing marked injection of the vessels and hemorrhage invading the cortex for a short distance. The cortical tissue otherwise is negative.

finding of the organisms make it much more probable that the monkey died from bacterial infection than from encephalitis. Flexner reported inconclusive results; Strauss, Hirshfeld and Loewe have published a preliminary report that indicates that the disease may be reproduced in monkeys by inoculation. I regret that up to the present time the lack of monkeys has made it impossible for us to do any work along this line at the research laboratory.

PATHOLOGY

Lethargic encephalitis belongs to the class of inflammatory diseases, in which also are included poliomyelitis, syphilitic lesions of the central nervous system and trypanosomiasis. While these different diseases have, broadly speaking, certain characteristics, the cases in a given class differ so widely that it is difficult, if not impossible, to accurately diagnose, by a study of the pathology alone, the less typical cases.



Fig. 3.—Cord, just below the olivary body, showing dense perivascular, round-celled infiltration and edema.

The meninges are usually described as showing only slight changes—an increase in the cellular elements particularly in the neighborhood of the blood vessels of the pia-arachnoid.

The cerebral cortex is generally normal except for congestion of the vessel of the leptomeninges.

In the brain substance, the changes are most marked in the basal nuclei of the brain, the upper part of the pons and peduncles, the gray matter of the floor of the fourth ventricle, and the aqueduct of

Sylvius. The changes in the medulla and cord are often reported as less pronounced, though observers have noted the same changes occurring in the upper section of the cord. This was certainly observed in the case of 48, which came to necropsy. To the localization in the mesencephalon, particularly in the vicinity of the nucleus of the third nerve, McNalty attributes the stupor, since a lesion in this locality cuts off the afferent stimuli. The relation of the paths of the rubro-

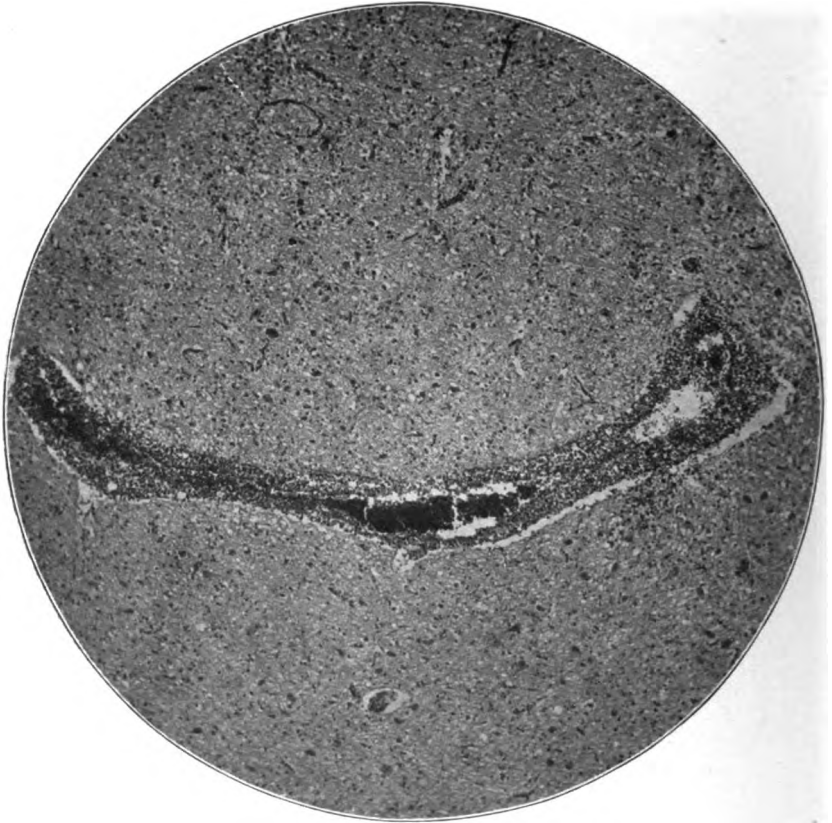


Fig. 4.—Cord, a higher level, showing dense perivascular and also diffuse round-celled infiltration.

spinal and pyramidal tracts to the region of the nucleus of the third nerve also explains the tremor and the frequent presence of the Babinski sign. It must be borne in mind that a virus affecting the nervous tissue, although it may have a predilection for a certain part of the central nervous system, may attack any part.

The lesions are generally described as consisting of four kinds:

1. Infiltration of the walls of the small vessels with lymphocytes and plasma cells.

2. Foci of interstitial and parenchymatous infiltration with round cells. In this reaction neuroglia cells may take part.

3. Lesions of the nerve cells—usually not so extensive as in poliomyelitis, and with less neuronophagia. These lesions of the cells usually occur when the inflammatory process takes place in the gray matter, but they may develop in the absence of any inflammatory reaction. Such is the case with regard to the cells of Purkinje in the cerebellum where inflammatory changes are almost entirely absent.

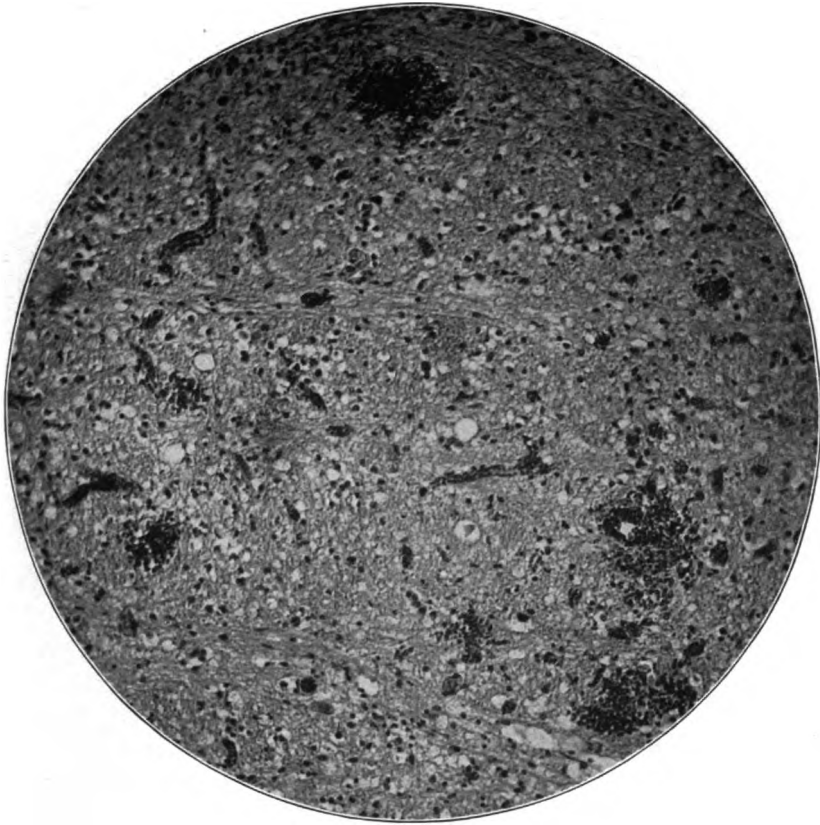


Fig. 5.—Medulla oblongata showing numerous petechial hemorrhages, diffuse round-celled infiltration and degeneration of ganglion cells.

4. Foci of perivascular hemorrhage. The vessel walls are usually not necrosed.

In connection with the statement that lesions of the cells may occur in regions where there is no evidence of inflammatory reaction, it is interesting to recall that Abramson, in a very excellent study of the pathology of poliomyelitis made at the research laboratory during the epidemic of 1916, brought out the same fact in regard to the lesions of poliomyelitis.

Perhaps an idea of the pathologic picture may best be obtained by a description of a case, No. 48. The brain was studied at necropsy by Dr. Alexander Fraser of Bellevue Medical School, to whom I am indebted for the following report.

MACROSCOPIC

The pia-arachnoid of the whole brain, including the medulla oblongata and a small part of the spinal cord accompanying it, shows

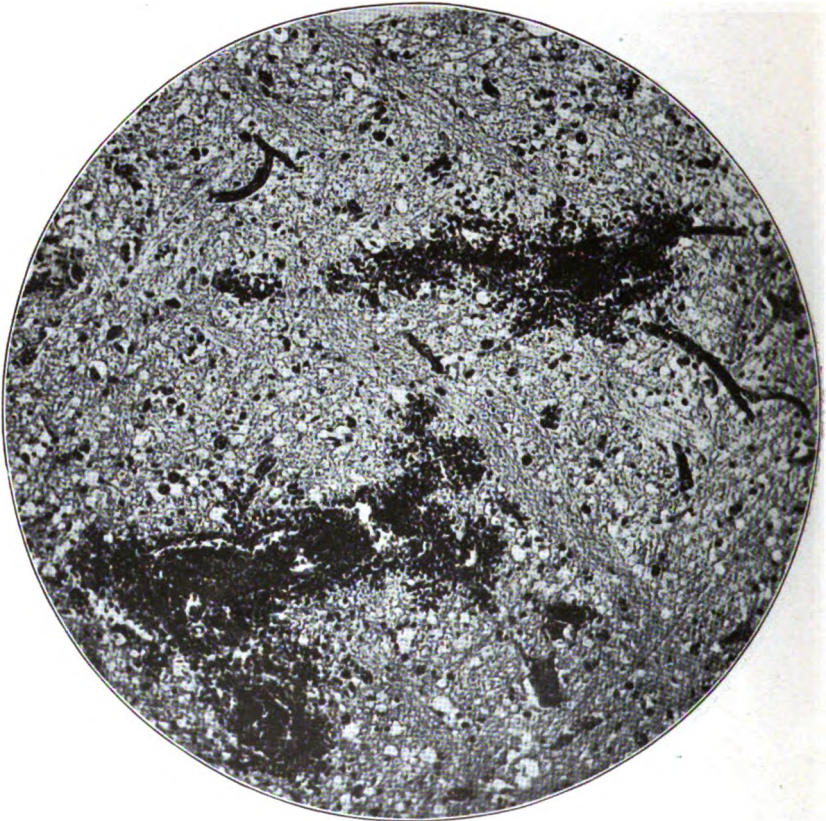


Fig. 6.—Another section from the medulla showing larger hemorrhages.

marked congestion of the vessels and numerous small hemorrhages. The portion covering the medulla, pons and peduncles is considerably thickened and brownish-gray in color. No free exudate is present. The brain tissue is firm in consistency. On section, the cerebral hemispheres show considerable distention of the vessels with blood and an occasional small splotchy hemorrhage especially in the outer cortex.

The ventricles seem large, but contain little fluid which is of a reddish tinge. The ependyma, especially over the thalamus and floor

of the fourth ventricle is lustreless, dull grayish-white in color and "mushy." In one place in the fourth ventricle it projects into the cavity in the form of a polypoid mass. The choroid plexus of the lateral ventricles is markedly congested and in places cystic. Section of the cerebellum shows distended vessels but apparently no hemorrhages. Section of the medulla, pons, crura and basal ganglions shows very marked distention of the vessels with numerous small and a few fairly large, irregularly outlined extravasation of blood.



Fig. 7.—Section of the cord showing central canal. Note disintegration of the epithelium and exudate in the lumen.

The color of the tissue in these regions is a fine and irregular mottling of dull gray and white. These features are especially marked just beneath the floor of the fourth ventricle.

MICROSCOPIC

The pia of the cerebrum and cerebellum shows distention of the vessels with blood, round cell infiltration especially marked around and

in the vessel walls, thrombosis and hemorrhages. Only very rarely does the perivascular infiltration follow the vessels into the brain substance, and then only for a short distance. Occasionally, a hemorrhage from one of these vessels is seen in the outer part of the cortex. In such areas the ganglion cells show various degrees of degeneration.

In the medulla, pons and basal ganglions, the pia shows the same pathologic features, but in a much more marked degree. In these

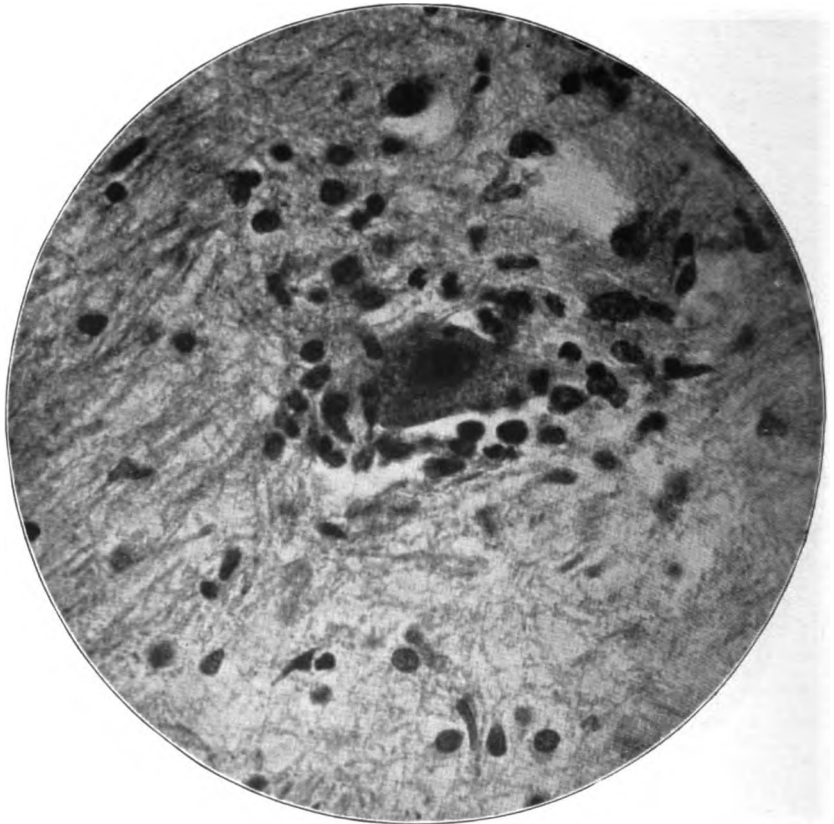


Fig. 8.—Same section as Fig. 7. Ganglion cell undergoing disintegration surrounded by phagocytes.

regions, too, the perivascular infiltration follows the vessels deeply into the brain tissue, and focal and diffuse areas of round cell infiltration are scattered throughout the tissue apart from the vessels. Frequent small and occasional large extravasations of blood are seen anywhere, but especially in the gray matter.

The cellular infiltrations, too, are practically always in the gray matter. The cells of the infiltrate are mostly lymphocytes with a few plasma cells and an occasional large mononuclear.

The blood vessels, though densely infiltrated, show no endarteritis.

In the affected areas, ganglion cells are seen in all stages of disintegration, some having been completely destroyed and replaced by groups of phagocytes. Sections stained by the Levaditi method were negative.

CONCLUSIONS

1. The histologic picture of the condition is that described by English authors for lethargic encephalitis, also that of trypanosomiasis.
2. The picture is very much like poliomyelitis, but such extensive infiltration of meninges and larger vessel walls would, at least, be very unusual.
3. The picture is also very much like syphilis. In syphilis, however, the infiltration sticks to the vessels. Syphilis shows an endarteritis, and usually gummata in vessel walls.

POSSIBLE RELATION TO POLIOMYELITIS AND INFLUENZA

Three theories have been advanced to explain the occurrence of lethargic encephalitis. When it first appeared in England, it was suggested that it was caused by food — botulism or some poison derived from substitutes or solanin accumulating in sprouts of potatoes or other vegetables. This theory has been definitely disproved and discarded. According to a second theory, it is a form of poliomyelitis; and, according to a third, it is connected with the epidemic of influenza.

The theory that it is a form of poliomyelitis has not been definitely proved or disproved. Epidemic poliomyelitis usually occurs in hot weather, the majority of the victims are children, and the lower motor neuron type of paralysis constitutes the great majority of the cases with paralysis. The onset is usually sudden and the greatest number of deaths occur in the first week. Lethargic encephalitis has occurred in its present appearance during the cool weather, the majority of cases having been adults (my own list of cases shows a large number of children, but this is undoubtedly because I am so often called to see the milder type of case where tuberculous meningitis is suspected). Very few cases of the lower motor neuron type of poliomyelitis are occurring, and among the cases diagnosed as lethargic encephalitis there are evidences of involvement of the higher centers in the way of cranial nerve palsies and prolonged lethargy which are rare even in epidemics of poliomyelitis. Certainly among the hundreds of cases of the latter disease that came under my observation during the epidemic of 1916 there were no cases at all approaching the characteristic pic-

ture of lethargic encephalitis, and only a few of the encephalitic type of poliomyelitis, with which some of these milder cases might easily be confused. Moreover, in lethargic encephalitis the onset is usually slow, and death occurs oftenest in the third week. The similarity of the spinal fluid findings is of little significance, since in neither case are they specific. The same may be said in regard to the pathology, though here there are, as a rule, more points of difference. As regards animal inoculation, it is certainly much more difficult to reproduce the disease in monkeys than is the case in poliomyelitis. For all these reasons it seems to me probable that lethargic encephalitis is not a form of poliomyelitis though the causative agents in the two diseases may perhaps be closely allied.

In regard to the possible relation between influenza and lethargic encephalitis, the evidence is as yet entirely circumstantial. In the first place, attention may be called to the fact brought out by historical study that on several occasions epidemics of a disease resembling lethargic encephalitis and influenza have occurred together. The impression is gained from these studies that encephalitis has not appeared in anything like an epidemic form except with influenza. It is certain that in their last appearance, 1889-1890, they occurred simultaneously, and it would seem that enough time has elapsed since for either to appear by itself if there were no direct connections between them. Then again, in a large proportion of cases, occurring in this country at least, the onset has been preceded by an attack clinically influenza. Moreover, that influenza has a marked effect on the central nervous system is shown in two ways: First, in nearly every instance, the convalescence from influenza is characterized by a profound mental depression and nervous exhaustion out of all proportion to the severity of the disease; secondly, as indicated by the reports of Jelliffe, Menninger, Burr and others, influenza is far more likely than any other acute infection to be followed by disturbances of the nervous system either psychic or organic. Therefore, it seems to me probable that there is a definite connection between influenza and lethargic encephalitis. Just what the relation is, I am not prepared to state. In view of the fact that we do not know the cause of either disease, one cannot say that the two diseases have the same origin. The causative agents may be identical or closely allied or the virus causing influenza may make the individual more susceptible to the causative agent of encephalitis or it may enhance its virulence. It is easy to speculate on the unknown.

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DISCUSSION

DR. JOHN F. HOGAN, Baltimore: In Baltimore we wished to ascertain the number of cases of encephalitis lethargica, as only a few were diagnosed. I saw all of the cases reported to the health department and there was one constant symptom or rather complication. I think it should be classed as a complication rather than a symptom, because it comes on later in the progress of the disease. This constant complication of diplopia seemed to set in after-

ward, within three to five weeks after the onset. In all the cases that I saw, the complication was present or developed later. The first patient I saw was a big husky man, 20 years of age. I did not know what his trouble was. He had been sent to a camp in that vicinity because he was a member of the Dental Reserve Corps. In February or March I heard that this man had recovered. He had had a slight facial paralysis which afterward disappeared, and as I questioned him and read the article to him, which appeared about that time in the report of the Surgeon-General of the U. S. Public Health Service, and I described diplopia he said he had it. He spoke of attending a lecture at the camp and while at the lecture he thought he saw two lecturers on the platform and, in fact, questioned a soldier who was next to him and asked him if there were two persons on the platform. I wonder if others had the same experience.

PSYCHOSES ASSOCIATED WITH INFLUENZA

II. SPECIFIC DATA. AN EXPOSITORY ANALYSIS *

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Psychic disturbances associated with influenza are first mentioned in the literature in connection with an epidemic of 1385 in Germany.¹ The "deliria" of the epidemic of 1387 are mentioned in accounts² by Valescus de Taranta and Gassar. The "vexatious deliria" of the epidemic of 1510 are mentioned by Mezèray.³ In "Annals of Influenza,"⁴ published in 1852, Riverius⁵ is quoted regarding the epidemic of 1580 thus: "It began with a fever and cough, then followed again a pain of the head and loins, then the fever intermitted a few days and returned with fresh vigor. Some had no rest, but the heat increasing they died; as some did of a phrenzy (!) and others of a consumption." Henisch the First spoke of the extreme prostration, "somnolent states, lipothymias, and other disquieting incidents" of this same epidemic. Quoted by Espagnol,⁶ Ozanam⁷ recounts the occurrence of "such grave symptoms as convulsive movements and somnolence" after the disease in the epidemic of 1691. Schweig⁸ described an epidemic in 1737 with psychic disorders similar to those he found mentioned in connection "Maladie du Sommeil," because of the somnolent and stuporous states following it (1718).

* Contribution I was published in J. A. M. A. 72:235 (Jan. 25) 1919.

* A contribution from the Psychopathic Department, Boston State Hospital series of 1919.

1. Schnurrer: Chron. der Seuchen, t. I, par. 352; 1385.

2. Kusnekow, A. C., and Hermann, F. L.: Influenza, Eine geschichtliche und klinische Studie, Wien., 1890.

3. Mezèray is quoted by Leledy but no reference could be found except to Sennert, de Abditis Rerum causis, lib. II, cap. 12, 510, q. v.

4. Annals of Influenza in Great Britain from 1510 to 1837; Thompson, London, 1852.

5. Riverius: Opera omni medica, Lugd. 1669, p. 585.

with the epidemic of 1580. Camerarius⁹ gave influenza the name

6. Espagnol, Jean: Contribution a l'etude des rapports de la grippe et du systeme nerveux, Toulouse, 1895.

7. Ozanam: Histoire médicale des maladies épidémiques contagieuses et épizootiques, Paris, 1835.

8. Schweig: Die Influenza, Berlin, 1836.

9. Camerarius: Quoted by Espagnol, without reference.

But despite these and other mentions of the concomitant deliria and numerous neurological manifestations, it remained for an American to signalize the association of "symptoms of madness" and "loss of reason" with influenza. This was Benjamin Rush of Philadelphia, in "An Account of the Influenza as it Appeared in Philadelphia, 1789, 1790 and 1791."¹⁰ Thereafter there were sporadic reports, for example, by Bonnet in 1837 "of an individual in whom the grippe provoked a furious mania"; by Pétrequin, who spoke of "patients tormented by sad ideas," and of "four or five suicides . . . in the hospitals of Paris" in the epidemic of 1837; by Chrichton-Brown in 1874, of "acute dementia following the grippe."^{11, 12, 13}

The epidemic of 1890 was evidently followed by many cases of mental disease, and the increased interest in psychiatry at this period brought forth a shower of literary progeny. Innumerable articles and reports appeared then and thereafter relative to the association of influenza with mental and nervous disease. Of the hundreds of names which are available in any complete bibliography, a few stand out as of preeminent importance and are still frequently quoted. Thus Kraepelin¹⁴ and Bonhoeffer¹⁵ are known because of their later prestige; Leichtenstern¹⁶ for his comprehensive study of influenza as a whole; Kirn¹⁷ because of his numerous able contributions; Ladame,¹⁸ Bidon,¹⁹ and others of the French school because of the different viewpoint represented by them; and Leledy²⁰ and Bossers²¹ for thorough historical and bibliographical records of the association of influenza and

10. Rush: *An Account of Influenza as It Appeared in Philadelphia, 1789, 1790 and 1791. Inquiries and Observations*, Philadelphia, 1805, p. 353.

11. Bonnet: *J. de méd. de Bordeaux*, 2nd series, Part 5, p. 175, 1837.

12. Pétrequin: *Gaz. méd. de Par.* **51**:801 (Dec. 23) 1837.

13. Chrichton-Brown, J.: *West Riding Lunatic Asylum Medical Reports* **55**:269, 1874.

14. Kraepelin, E.: *Ueber den Einfluss acuter Krankheiten auf die Entstehung von Geisteskrankheiten*, *Arch. f. Psychiat.* **11**: 1881, and **12**: 1882. Also, *Ueber Psychosen nach Influenza in Dorpat*, *Deutsch. med. Wchnschr.* **11**: (March 13) 1890.

15. Bonhoeffer: In *Aschaffenburg's Handbuch* (Leipzig and Vienna), 1912.

16. Leichtenstern, O.: In *Nothnagel's Encyclopedia*. "Influenza." English translation by Stengel, 1905, Philadelphia, W. B. Saunders Co.

17. Kirn: *Ueber Influenza Psychosen*, *Munchen. med. Wchnschr.* **37**:299-301, 1890. *Die nervoesen und psychischen Stoeurungen der Influenza*, *Samml. klin. Vortr.*, n. F. **23**: Leipzig, 1891; *Die Psychosen der Influenza*, *Allg. Ztschr. f. Psychiat.* **48**:1-15, 1891-1892.

18. Ladame: *Annal. méd.-psychol.* **13**:20, 1896.

19. Bidon: *Revue de Médecine*, 1890.

20. Leledy, A.: *Lagrippe et alienation mentale*, 4. Paris, 1891.

21. Bossers, A. J.: *Die Geschichte der Influenza und ihre nervoesen und psychischen Nachkrankheiten*, Leiden, 1894.

psychosis. (The historical summary of the first paragraph of this paper is based in large part on these accounts.) In addition there were a great number of current literature accounts — about 220 authors are listed in the Surgeon-General's Index Catalog, 1902 volume. Books on the subject were issued in German by Ahrens, Borchardt, Bossers, Brachmann, Eberlings, Hirschfeld, Jutroinski, Klemm, Lochner, Mispelbaum, Mucha, Wescher and Weynerowski; in French by Auguin, Biet, Brionne, De Groote, Diemer, Espagnol, Lehmann, Le Joubieux, Leledy, Lestra, Ulliel, Trastour, and Virey; in Danish by Fehr, and in Italian by Rossi. I know of no books in the English language.

All of these accounts have a certain intrinsic interest, but many of them are of dubious value to the present day psychiatrist because of the ambiguity of the antiquated terminology. The insistence on interpretation instead of pure description also militates against the usefulness of many of the case histories, and the original meanings of many of the terms used have now become obsolete. This may be illustrated by the extreme elasticity and vagueness, even today, of the protean term "delirium."

THEORY OF THE SPECIFICITY OF MENTAL SYNDROMES

Since 1890 the reorganization of nosologic classification, the animadversions of Freudianism and psycho-analysis, and the freedom from pandemics have combined to keep literature on the mental side of influenza almost negligible in amount. There have been a few contributions, such as that of Gosline,²² Jelliffe²³ and others, but none has made any claim to exhaustiveness. There has been, on the other hand, rather a tendency to generalization and to the assigning of categorical principles even before the complete assembling of pertinent details. Thus arose the question of the specificity of mental syndromes with acute infectious diseases, ardently sponsored by Kraepelin and vigorously combated by Bonhoeffer and the French school. Kraepelin's prestige was sufficient to project, even in the face of majority opposition, the thesis that each acute infectious disease could, in the course of further study, be recognized from the mental symptoms alone. This, in short, is equivalent to declaring for the autonomous entity of a variolar psychosis, a typhoidal psychosis, an influenzal psychosis, etc. Except in the case of pellagra (an exceedingly dubious exception), this possibility is not yet recognized, nor is there any general tendency to ascribe the fact that it is not to our failure to read all the signs, as Kraepelin maintained.

22. Gosline: *Newer Conceptions of Dementia Praecox*, J. Lab. & Clin. Med. 2:691 (July) 1917.

23. Jelliffe, S. E.: *Influenza and the Nervous System*, Phil. Med. J. 1041 (Dec. 27) 1902; *Psychoses with Influenza*, N. Y. Med. J. 108:725 (Oct. 26) 1918.

It cannot be gainsaid, however, that influenza apparently produces an exceptionally potent neurotoxin. After the last great epidemic, in 1890, Leichtenstern¹⁶ in his celebrated monograph wrote "This epidemic has taught us something quite new in neurology, namely, an acute infectious disease which compared with all others is characterized by its striking neurotoxic effects. The sentence . . . 'The influenza toxins are intense nervous poisons' has found full justification in the history of earlier as well as in the latest epidemics." Most writers agree with Leichtenstern that influenza is preeminent as an inciter of nervous system sequelae, although Bonhoeffer¹⁵ and a few others believe in the supremacy of typhoid. But even to the present time there is no proof that these sequelae are qualitatively specific.

THE QUESTION OF PREDISPOSITION

A corollary question arose which is in a sense more important. This is the question of nervous predisposition as a soil for the psychopathies and neuropathies which arise after influenza. It was Leichtenstern's opinion that "influenza produces a specific nervous toxin which, besides producing conspicuous nervous prostration, in some cases, by its poisonous action on the cortex of the brain, calls forth these psychoses." He points in proof to the two facts, that (1) "the cases observed by us were conspicuous by the fact that hereditary or other neuropathic influences were not found in them, and (2) the obvious frequency with which the young, even infants, were affected by these psychoses." Opposing this view were many writers: Ladame, Mehr, Mispelbaum, Ayer, Justrosinski, Kraepelin, et al. Kraepelin took herein a rather self-contradictory view, for despite his generic theory of specificity, he declared in regard to this particular disease that "it would seem that influenza alone would not be able to produce a disturbance of the mental function in a normally constituted person." Schuele echoes this: "Influenza only plays the part of an etiologic accompaniment in the production of mental disease." And Ladame, "Influenza by itself is never sufficient to produce insanity." Leichtenstern marshals Althaus, Bossers, Mucha and Ulliel as authorities admitting of the possibility of psychoses arising from influenza alone, without "nervous predisposition," and quotes Kirn as having found evidences of predisposition in only 23 per cent. of the *cum-febrile* psychoses, but in 92 per cent. of the *postfebrile* psychoses. He attacks the evidences for predisposition in the latter, however, and, to the writer, seems justified.

At present these questions remain much as at the time of this discussion twenty years ago. The recent pandemic of influenza stands in a fair way to settle them. Of the important acute, infectious dis-

eases occurring in epidemics, typhoid and variola are so well controlled that any large mass of material from which to extract psychiatric data is not to be expected. Plague has not of recent years been epidemic, and the present advance of preventive medicine is not likely to permit it to become widespread. Influenza alone remains, and no comment is necessary to remind us of its ubiquity.

THE BOSTON SERIES

About 120 cases of mental disease with a history of recent influenza were admitted to the Boston Psychopathic Hospital during the last three and one-half months of 1918. (About sixty additional cases were seen early in 1919, and will be presented in future communications.) A statistical analysis of eighty of this group was recently presented²⁴ as the first of a series of papers dealing with the subject from this hospital. I pointed out then that on the basis of statistical indications, the prominent points brought out were the frequency of schizophrenia (*dementia praecox*) as a postinfluenzal psychosis, the difficulty in diagnosis between delirium and schizophrenia in many cases, the wide variety of psychotic syndromes observed and the conspicuous absence of stigmata of mental predisposition in the majority of the cases.

The answers to the two questions of specificity and of predisposition are thus strongly suggested. But these answers may not be learned from data purely statistical. For one thing, they fail to consider certain essential mathematical fallacies. Thus, 10 per cent. of cases observed in this hospital may have been deliria, but this in no accurate way represents the relative frequency of delirium in influenza or its frequency as a form of mental syndrome accompanying influenza.

Consequently, further study of the data accumulated is expedient, and is herein set forth. Of various modes of attack on the problem of "What forms of mental disease result from influenza?" an expository analysis by case exemplification lends itself with particular facility. The 120 cases of psychoses in the production of which influenza was an apparent factor, embrace types of all the recognized psychiatric groups, and the data of typical cases constitute the most accessible and forceful negation at hand of the theories of specificity and essential predisposition. The variety of psychiatric manifestations and the varying rôles of influenza, primary, secondary and collateral, appear from the following representative cases.

24. Menninger, Karl A.: *Psychoses Associated with Influenza*. I. A Statistical Analysis, *J. A. M. A.* **72**:235 (Jan 25) 1919.

In the presentation of these I have chosen to follow the classification of Southard.²⁵ His regrouping of the major forms of mental disease into eleven large categories has the great advantage of convenience. This virtue appears in a comparison of the facility of illustrating the main types of mental disease encountered in the present series. For while it would be quite feasible to illustrate at least twenty of the twenty-two of the psychoses of the American Medico-Psychological Association list (pellagra psychosis, for example, omitted) with influenzal psychoses, it would in no way amplify, except in bulk, the contribution of an illustration of each of the eleven groups of Southard. Without further comment, these appear.

ILLUSTRATIVE CASES

GROUP 1.—*Syphilopsychoses. Neurosyphilis*

The precipitation of the psychoses of neurosyphilis by acute infections is well known because of the spectacular results rather than because of its frequency. There is a surprising paucity of references to it in the literature, however. The following is one of a series of this type.²⁶

CASE A.—*Latent Neurosyphilis + Influenza = Active Paresis.*

Family History.—The patient was an unmarried man, aged 46, a watchman by occupation, born in the United States. The family history was entirely negative, including an account of the grandparents, uncles, aunts and siblings, except that there were three siblings who died at birth.

Past History.—He had evidently been a healthy, normal child, and graduated from grammar school at 14, entering economic life as a shoe-store clerk. Subsequently he worked in the city fire department. At the age of 32 his thigh and hip were fractured in an accident, and he was given a position as customs guard. He worked steadily until the day he went to bed with influenza. There is no court record. He used no alcohol and no tobacco. Except for the fractured hip his medical history is negative. In disposition he is described as being happy and sociable, with a fondness for music and the theater. The only previous symptom of any kind which was elicited was that he had complained of being tired during the past four summers.

Present Illness.—September 20, he contracted influenza, which evidently attained a rather severe degree. At one time the temperature was reported as 104 F.; but returned to normal on the fourth day. He arose from his bed and was up and about the house, planning to return to work, but seemed to contract "a cold" again and returned to bed two weeks after arising. He was in bed part of the day, but was up the next day, feeling quite well again. That night he became "delirious." He is described as having been irritable and having refused to take the medicine from the doctor, declaring that it

25. Southard, E. E.: The Genera in Certain Great Groups or Orders of Mental Disease, *Arch. Neurol. & Psychiat.* 1:95 (Jan.) 1919.

26. Menninger, K. A.: Influenza and Neurosyphilis, *Arch. Int. Med.* 24:98 (July 15) 1919.

was the wrong kind. He hung his head over one side of the bed and let it so remain. He refused to do anything he was told, and announced that the Blessed Virgin Mary was going to tell him what to do. About this time occurred, according to his wife, one—possibly two—brief seizures. He was brought to this hospital, the admission not provoking any severe psychic reaction.

Mental Examination.—(Summarized.) His general attitude was that of accessibility and cooperation, although he was at times self-absorbed and agitated, or depressed. His *sensorium* seemed to be entirely clear. His *orientation* was precise, except that he had no memory of how long he had been in the hospital. His *memory* was grossly defective. Not only were many facts of his past history forgotten, but details of all kinds were lost, and his memory for recent events was extremely poor. He recognized short periods of total amnesia occurring during the previous two weeks. His ideation showed fleeting persecutory delusions without systematization, and certain delusions of other types—ideas that his mother was dying, that the Virgin Mary would guard him, that misfortune was about to overtake his family. No hallucinations were elicited. His *emotional tone* showed, as stated above, instability and, at times, a tendency toward mild, agitated depression; he frequently wept. The *thought processes* were badly disrupted, but there was no blocking, retardation or acceleration. His *motor activity* was slightly decreased.

Physical Examination.—This showed the patient to be a poorly nourished, poorly developed man, aged 46, 5 feet, 7 inches tall, weighing 130 pounds. Special senses, epicritic and protopathic sensory interpretations were normal; but deep sensation somewhat impaired. There was no Babinski and no ankle clonus, but a slight swaying in Romberg's position. Knee jerks were equal and active. Pupils were contracted, fixed, and reacted neither to light nor to distance. Corneal and pharyngeal reflexes were present; extra-ocular movements, normal; abdominal and cremasteric reflexes, absent. There was tremor of facial muscles, tongue and extended hands.

Laboratory Findings.—The urinalysis was negative. Blood serum Wassermann test, positive. Spinal Fluid: Albumin, ++; globulin, ++; cells, 41; colloidal gold reaction, 5555555555. Wassermann test, positive.

Diagnosis.—General paresis. He was committed.

GROUP 2.—*Hypophrenoses. Hypophrenia*

No positive proof in confirmation of the production of Kraepelin's dubious "infectious idiocy" in previously sound material was obtained. This term is a glaring example of Kraepelin's inaptness at nomenclature, as the expression "infectious idiocy" certainly violates all canons of correct designation of mental disease. "Infection dementia," while open to criticism, is certainly more accurately expressive of his meaning. Waiving logomachical contentions, however, we may say that there has been no present evidence of an utter annihilation of mental processes by influenza in nonfatal cases.

Subtotal mental or intellectual loss was, however, definitely demonstrated. The following remarkable case is one of several of a similar

nature observed. Burr has mentioned having seen several such in a recent report.²⁷

CASE B.—Morosis + Influenza = Imbecility.

Family History.—The patient was a boy aged 10, of American parentage. The family history is entirely negative. An only brother, a year younger, is quite well, and is doing fair work in school; he is in the fifth grade.

Past History.—He was born at full term—normal delivery, and had had no severe illnesses. His teeth appeared at 5 months; he walked at 18 months; talked at 3 years, and started kindergarten at 4.

School Record: (His teacher was interviewed.) He did poor work in kindergarten the first year and was kept there a second year, which is only done "with particularly backward children." He was promoted to first grade, but had to repeat the work, and then, though it was still poor, he was promoted. "His work in second grade was poor but he was promoted because considered too old to stay in the grade." The third grade work was poor, and was repeated this year . . . record, so far, poor. The patient had so far had the very lowest rank given, and was promoted only because of lack of better arrangement, and because of a desire not to keep backward children behind indefinitely.

Disposition: He was always quiet, rather seclusive and played with childish toys which even the younger brother has outgrown. His teacher reports that his deportment is good, and that he does not seem sensitive about his backwardness. There had never been any conduct disorder of note.

Present Illness.—He had influenza for four days, temperature not known. He "slept without waking" for three days, taking no food. The physician and his nurse insisted on his staying in bed a week, which he did without complaint, "perfectly normal and happy" up to this time. Three weeks after the influenza (on Christmas morning) he complained of headache and fatigue, and by his parents' statement "has been out of his head since." (The history was obtained on the 28th.) He cried constantly; wandered aimlessly about; went outdoors and became lost; occasionally sang in a feeble voice numerous popular songs; at other times screamed and cried and refused to remain in bed; said, "I'm lost. Take me home. I will be good to you." His parents were most alarmed because he failed to recognize his mother. He was brought to the outpatient department and recommended to the house.

Mental Examination.—For the first two days he was constantly blubbering and wailing, the more so when any attempt was made to comfort or quiet him. He selected one of the nurses finally, whose attentions he would permit, and dragged himself about the ward at her heels thereafter, crying when any one else approached him. He did not scream petulantly, but cried as if in grief and begged to be taken home to his mother. He did not express delusions or hallucinations. He was sleepless for the greater part of the first four nights, but gradually slept better at night and cried less in the daytime until, on the fifth day, he was quiet all day, clothed, accessible, and, in a faint way, cooperative. *Orientation* for place, time and person was retained from the start. His *memory* defect was most prominently shown in school learning, his knowledge of more general matters being more open to question than his memory thereof. He was tested by the tests used at the Waverley

27. Burr: Mental Complications and Sequelae of Influenza, *Med. Clin. of N. A.* 2:709 (Nov.) 1918.

School for the Feeble-minded and found incapable of doing any of the third grade tests, many of the second grade tests, and not a few of the first grade requirements. Thus, to select some of the most glaring deficiencies, he could not count by twos, he could do no subtraction at all, he misspelled "do," "go," "run" and other words little more difficult. He did spell correctly "cat," "it," "and," and "an," and did a few simple problems in addition, such as "2 + 3, 1 + 4, and even 3 + 7," but he could not get much above the latter. *Ideation* was represented by an absence of delusions and an absence of insight. He gave a very inadequate account of his recent trouble, although he did not seem to have had any amnesic periods. *Hallucinations* were not proved. *Thought processes* showed no defect of attention after the fourth day; the associations which were rather slow were not schizophrenic, incoherent or erratic, although of course quite puerile. *Emotional tone* was, of course, a point in question. He was very lonely and nostalgic at first; whether or not this accounted in any considerable measure for his conduct is questionable. During the examination he showed no emotion except when home was mentioned, which evoked a few quiet tears. On the day of discharge he was quite elated and happy because he was going home. *Motor Status*.—His conduct disorder was confined to the weeping and wailing and screaming of the first few days.

Subsequent History.—On the day of discharge—January 4—his lugubriousness was replaced with smiles and complacency. His memory was again tested briefly and found unimpaired, even for the recent episode; insight, essentially lacking, although he did say that his head was "not right" and that he had been "dizzy from rocking too much;" intellectual processes, definitely, but slightly improved; ideation, as before, and conduct, above reproach. But he took no interest in trying to do ward work nor gave other manifestations of normal initiative.

Physical Examination.—He was a rather undersized lad whose physical examination was negative in all respects save for "a suggestion of hutchinsonian teeth." These were the incisors, particularly the uppers, which had an elliptical or concave contour of the margin, not typically hutchinsonian.

Laboratory Findings.—Urine negative. Blood serum Wassermann test negative. Spinal fluid negative, including Wassermann test.

Psychometric Tests.—The patient graded regularly, variation total of 7, at a mental age of 5.7 on the Yerkes-Bridges Point Scale. On the Stanford Scale, four days later, he graded at 6 years, 2 months. In the supplementary tests his performance in the construction puzzles was poor. The memory tests were also poorly done, and he accepted ten out of ten suggestions. The patient cooperated poorly on the Point Scale and fairly well on the Stanford.

He was discharged with recommendation for instruction in a school for the feeble-minded. The diagnosis at discharge was imbecility.

GROUP 3.—*Epileptoses. Epileptic Psychoses*

The final word on the influence of influenza on the production or augmentation of epilepsy should come from the institutions primarily devoted to the care of that disease. The first mention of the matter is probably that of Michell,²⁸ who wrote of the epidemic in Holland, 1782,

28. Michell: Antwood over de Febres catarrhales; in *Verhandelingen van het Zeeuwsch Genootschap der Wetenschappen te Vlissingen*, p. 162; Michell: *Geneeskundige Verhandeling over de . . . Febres catarrhales . . . in den Nederlanden, Middelburg, 1875.*

that it "brings about in the brain apoplexy, epilepsy, chorea and . . . convulsive movements." The direct causation of "a typical but chronic epilepsy" which, however, always recovered, has been reported by Landgraf,²⁹ Van Deventer,³⁰ Jaccoud³¹ and Leichtenstern.^{16, 32} Of this type we saw none whatever. Our experience has been quite contrariwise, that epileptic attacks were indeed precipitated or accelerated in known epileptics, but that in no unpredisposed case did they occur for the first time after influenza. The cases of epilepsy, curiously enough, are so generally complicated by other factors, for example, hypophrenia, parturition and appendicitis, that it is difficult to present a good type case. The following case is interesting for the unusual change after influenza from the typical convulsive seizures to the somnambulistic or twilight-state episodes, and illustrates also an increase in frequency of attacks.

CASE C.—Occasional Epileptic Seizures + Influenza = Epileptic Twilight States and Increased Frequency.

Family History.—An unmarried American sailor, discharged, aged 22, had a negative family history except that one paternal uncle is said to have had epilepsy. A brother, aged 23, is living and well.

Past History.—The patient was born in Maine; had a normal childhood, and received a grammar and high school education. He left the high school to go to work, but later joined the U. S. Navy and attended a radio school. He was held back one year in high school because of delinquency in algebra and English. He enlisted in the U. S. Navy in April, 1917, and was discharged in March, 1918, presumably because of his epileptic attacks. He admitted having concealed his epileptic history on enlistment, but it was discovered in the Navy and he was held for medical examination in naval hospitals for some months before discharge. Since that time he has been employed at testing steel in an arsenal.

Medical History: This was negative except for epilepsy. He was never seriously ill nor injured. He used no alcohol, tobacco only moderately and denied sexual perversions.

Epileptic History.—Attacks began at the age of 12; no particular circumstances of the first seizure could be obtained. Thereafter they occurred about every two months until a few years prior to admission. The attacks entailed the characteristic tonic convulsions, frothing, biting the tongue, etc., but were not extremely severe and, as they were preceded by a somewhat prolonged aura, he had never injured himself severely. They were always followed by a period of headache and malaise, with total amnesia for the events of the seizure. For a year or so prior to his enlistment in the Navy they had been

29. Landgraf: Gesellschaft der Charité-Aertzte in Berlin, Berl. klin. Wchnschr. 1890, Nos. 9 and 12.

30. Van Deventer: Centralbl. f. Nerven- u. Psychiat. 13:49.

31. Jaccoud: Pathologie interne, Paris, 1870, p. 780, and Nouveau dictionnaire, etc., Paris, 1873, Part 16, p. 740.

32. Leichtenstern, O.: Influenza Lectures, publ. in Deutsch. med. Wchnschr., 1890, Nos. 11, 15, 18, 22, 23, 29, 30, 42, 43.

decreasing in frequency, under the treatment of Dr. Waterman of Boston, and during the period of his Navy enlistment, almost a year, he had but two seizures. He was discharged from the Navy in March and between then and November had two or three more attacks.

Present Illness.—September 30 he contracted influenza and was in the Homeopathic Hospital until November 1. He had pneumonia and was not expected to live. During his acute illness he was delirious and did not remember his father's visits. He had no epileptic attacks during the month.

During November and December he had three epileptic attacks. These were of the nature of somnambulisms, however, instead of the convulsive type as previously. He was first noticed by his employer one day to be managing his duties clumsily and when addressed did not reply; so he was taken home, where he subsequently recovered without memory of the event. On another occasion he suddenly left work, taking with him a friend's letter to which he was in no way entitled, and was observed to stagger as if drunk. The third attack was the event of his coming to this hospital. He last recalls being at his work as usual; the police found him wandering the streets in an adjacent suburb of Boston, and failing in satisfactory replies, he was brought by them here. "In the admission office he has to be prodded with questions frequently in order that any answers may be elicited, and to keep him awake. Said he could not tell where he has been living in Boston the past three years. Later replies that he was a radio student at Harvard."

He was taken to the ward, fell immediately asleep, and when seen the following morning was quite clear, but could give no account of the interval between leaving work the day before and awaking in this hospital.

Mental Examination.—This was negative. He was a well appearing, intelligent, alert young man fully accessible and cooperative. *Memory, orientation, ideation, thought processes, emotional tone and motor status* were quite normal; there were no hallucinations and no memory of any. He was amnesic for the periods of his epileptic attacks and for the periods during which he was delirious with the influenza and pneumonia.

Physical Examination.—Entirely negative.

Laboratory Findings.—Entirely negative, including spinal fluid and serum Wassermann tests. The *psychometric test* gave a rating of 17 plus.

Diagnosis.—Epilepsy, twilight state. Not psychotic.

The following case, while not one of the series observed at this hospital, is rather more representative of the general tendency here observed. This case was one seen at Dr. Myerson's clinic at the Boston Dispensary, where the writer was an assistant visiting neurologist.

CASE D.—Epilepsy + Influenza = Shower of Attacks, Then Complete (?) Cessation.

Family History.—A white schoolboy, aged 14, had a negative family history as far as frank epilepsy or other mental diseases were concerned. The father, aged 45, was subject to outbursts of temper, and the mother, aged 38, to chronic headaches, but such ubiquitous symptoms should probably be disregarded. One sister is living and well. Both parents were born in Ireland.

Past History.—The patient was born in Boston, 1904, and except for measles, rubella and chronic tendency to constipation, had always been well. He was in the seventh grade at 13, but lost ground because of his epilepsy and was repeating the grade at 14 when seen by us.

Epileptic History.—In August, 1916, he had a sudden epileptiform seizure which initiated his epileptic history. This and subsequent attacks were generally preceded by auras of dizziness and "stomachache," and an aggravation of his constipation generally foreran the attacks by several days, sometimes weeks. Just before the fall he was accustomed to see the walls and floor moving.

The attacks have occurred both day and night and in all sorts of places. He has, however, never severely hurt himself, bitten his tongue, or passed urine. He lost consciousness totally and had tonic and clonic convulsions. After the attacks, which are of variable duration, he has severe headaches. Occasionally the attacks are mild.

The average interval was given as "two or three months." As he was being treated in the diet clinic for constipation and in the skin clinic for herpes zoster, the attacks were usually noted, and the following dates appear, showing greater frequency: Oct. 10, 1917; Nov. 17, 1917; Jan. 30, 1918; "Is beginning constipated again"—April 24; May 2; June 23; July 15 \pm ; August 12.

Present Illness.—About the last week in September he contracted influenza, was very ill at the City Hospital and bedridden for a month. During his illness he had three, possibly (he himself says) four more seizures. The last was on October 21, while he was still bedridden. Thereafter he was seen in the nerve clinic frequently and had no more seizures. (Seen January 14; no further attacks up to that time.) A *physical examination* was entirely negative, and a Wassermann test on the blood serum was negative.

GROUP 4.—*Pharmacopsychoses.* Psychoses from drugs, alcohol, etc.

Paradigm: *Alcohol(ism) + Influenza = Delirium Tremens*

I recognize the criticism which the presentation as an influenzal process of so prosaic a phenomenon as suggested by this paradigm will provoke. Nevertheless, there is some defense. First of all, it is certainly the most frequent, and, secondly, it is by no means the best understood psychic manifestation of the combined effects of alcoholic and infectious toxemia. It is a considerable question whether the apparent is the real in the case of the suggestions of our paradigm. For all its plebeian frequency, delirium tremens seems far from being completely understood (and we have been awaiting the cry from the alcoholic business interests that the national prohibition amendment has foreclosed the possibility of further study of alcoholic psychoses in this country!). There are not a few (for example, Bonhoeffer) who do not regard the psychosis as a primary result of the alcoholic toxemia, or the direct effect of alcohol on the cortex. Nor is there adequate rationalization for both the excess and the withdrawal incidences.

In regard to the influence of influenza as a specific form of infection in the precipitation of delirium tremens, there are no conclusive data. Rosenbach³³ thought the epidemic of 1890 was followed by delirium

33. Rosenbach: Berl. klin. Wchnschr., 1890, p. 95.

tremens of usual frequency and gravity. But "the assumption that influenza on account of its marked neurotoxic character is relatively more frequently accompanied by delirium tremens than any other acute infectious disease, is quite unfounded. . . . The frequency of alcoholic delirium [sic] during influenza periods is noted on all sides. (Rosenbach, Nagy, Bruns and others) . . . (But this) is an absolute increase due to the enormous morbidity of influenza and especially to the frequent occurrence of influenza pneumonia." With these remarks of Leichtenstern¹⁶ we are in full agreement. The essential nature (and etiology) of delirium tremens is still incompletely understood.

Before proceeding to the details of the case to be cited, I wish to mention a particularly conglomerate case which is easily classifiable by means of Southard's grouping, but quite impossible to locate satisfactorily in the twenty-two titles of the A. M.-P. A. Manual. The patient was a successful surgeon, aged 38, with a family history of multiple instances of insanity on both paternal and maternal sides. He himself had long been alcoholic and had latterly become addicted to the use of morphin, hyoscin, and, possibly, of other drugs. This man contracted influenza and developed thereafter a psychosis as bewildering as the evident etiology, combining symptoms of paranoid schizophrenia and Korsakoff's syndrome. Incidentally, his father is thought to have been paretic, and the patient himself admitted syphilis, but the spinal fluid and the blood serum proved negative.

I will now review one of our typical cases of delirium tremens. Some of these psychoses began during the acute influenza attacks, some immediately afterward, some a few days after supposed recovery from influenza.

CASE E.—*Alcohol(ism) + Influenza = Delirium Tremens.*

Family History.—A bartender, single, aged 32, born in Massachusetts, had a negative family history.

Past History.—This was negative except for the following particulars: For sixteen years, more or less, he had been alcoholic, and especially so for the past three years. Until three years ago he worked as an egg inspector at \$18 a week, but later became a saloon porter and later a barkeeper. His own conservative (?) estimate of his average consumption during the last year or two was "two gallons of whisky and beer a day." He freely admitted having been "drunk" two or three times a week for the previous two years, but denied ever having been hallucinated; and he was not a solitary drinker.

Present Illness.—He entered the hospital with a history of having recently had influenza, subsequent to which he became noisy, excited and destructive in reaction to hallucinations. He entered in a hyperkinetic, euphoric state and "would rather sing 'On My Way to Mandalay,' than answer questions." He was *correctly oriented*, saying he thought this would be Friday "if it don't rain," and that he was now "in an Influenza Hospital" (which it might well have been taken for at that time). He remained quite disturbed, reacting to

many hallucinations, but without great conduct disorder until the fourth day, when he suddenly became clear and gave the following account of his present illness, which is established by comparison with outside history to be correct as to known facts.

He begins by asking if he has been in a trance all this time. Says he "just woke up this morning." Is not clear about recent events; thinks it was about the first of October (two weeks ago) that he took sick with influenza. (As a matter of fact it was four days prior to admission and eight days prior to the taking of the history.) He took to bed, and on the evening of the fourth day (he thinks the second) began to imagine foolish things, heard a band in the house, thought there were robbers in the building, saw the face of a robber in the window, saw "a little coon with a cigar in his mouth and a Quaker Oats box in his hands." The "robbers" remained about, too. He became very frightened, "yelled like hell" and scared the whole household. Threw the cuspidor at "the little coon." Kept the house awake. About 7 a. m. a doctor was called and found him "standing up, all in a sweat, much excited by all that had happened." The doctor sent him here. He remembers coming, being admitted, and being visited repeatedly by the examiner. Has a hazy memory of the whole affair, including many minor details, such as discovering that he had pediculosis pubis, etc.

Recalls also numerous delusions and hallucinations of the same period, and although they were real enough to him, he strangely does not confuse fact and fiction, although he says, "I couldn't swear to it." Some of these delusions and hallucinations were that he was in a saloon where he had once worked, and that a riot was in progress, that he was reprimanded by the "boss" for slack work, that there was a parade in which he was participating, that he was drunk, that a friend named Florence was hiding under a board, that an audience surrounded him, and that "rats, snakes, mice, scorpions and every kind of an animal except an elephant" were to be seen. These he thought he saw en route to the "Influenza Hospital." This morning he woke up and wondered where he was. Finally decided that he had been "in a trance for the past week." Thinks alcohol had much to do with it, and that he had "the shakes" the day he got sick with influenza.

Mental Examination.—After the disappearance of the acute visual and auditory hallucinations and concomitant delusions, no psychotic symptoms were observed. He remained quite clear after the fourth day and was discharged two days later with a diagnosis of delirium tremens, recovered.

Physical Examination.—This was entirely negative except for general tremulousness and a blood pressure of systolic 150, diastolic 105.

Psychologic Examination.—This showed a mental age of 11.5 years with a variation total of 15, "excellent" performance on the memory tests and an acceptance of only two of ten suggestions.

GROUP 5.—*Encephalopsychoses*. Psychoses with organic brain disease

Comment.—"Cerebral apoplexy has been described as a symptom of influenza in England as far back as the epidemic of 1743. . . . There are no accounts of any postmortem examinations in this period. . . . In 1890 . . . I first called attention to these apoplectiform hemiplegias and monoplegias occurring in influenza. . . . Anatomically there was . . . a focal acute hemorrhagic encephalitis."^{16, 29, 34}

34. Leichtenstern, O.: Primary Encephalitis, Deutsch. med. Wchnschr. 2, 1892.

Thus does Leichtenstern refer to this interesting phenomenon, influenza apoplexy which "must be regarded as a rarity." He cites cases reported, in addition to his own, by Virchow-Senator, Fuerbringer, Koenigsdorf, Schmidt, Erlenmeyer, Gross, Eichorst, Stembo, Remak, Drasche, Bilhaut, Prentis, Herzfeld, Warfvinge, Brakenridge, et al. Two cases reported by Straumann are cited which resemble the one here recorded "(fever, coma, death) . . . most readily classified under the . . . form of acute encephalitis without motor manifestations or motor paralyses." The pathology and a short discussion of the various forms is given, but he does not speak of definite psychoses accompanying or preceding the other manifestations, as herein-after related. Michell's²⁸ comment on the production of "apoplexy" by influenza has been quoted above. Oppenheim³⁵ refers in his textbook to his own work and that of Struempel, Lichtenstern and Fuerbringer, and gives a good description and photograph of the lesion. He does not discuss psychotic accompaniments.

Up to the present time the 1919 literature has contained no other reports of postinfluenzal hemiplegias (encephalitis hemorrhagica influenzae). In an address at the meeting of the Association of American Physicians at Atlantic City, June 16, 1919, Major J. W. Hall of Denver casually mentioned two cases seen by him in the course of his military medical work in the training camps of the southwest. In a personal conversation he stated that both cases were sudden hemiplegias occurring in young male adults during convalescence from influenza, one while straining at stool, and the other under circumstances not recalled. An effort is being made to collect details on these exceedingly valuable cases.

A few other instances of encephalopathic psychoses with an associated influenza were observed in our series, including one other instance of cerebral hemorrhage, but in a case psychiatrically diagnosed manic-depressive psychosis with good symptomatic basis.

It may be demonstrated later that encephalitis lethargica should be placed in this group of encephalopathies resulting from influenza. At the present time opinion on this point is sharply divided, and in order not to confuse the present issue, encephalitis lethargica (as opposed to encephalitis hemorrhagica) is not discussed or illustrated here.

CASE F.—*Normality + Influenza = Atypical Psychosis, Proceeding to Death from Cerebral Hemorrhages* (Hemorrhagic Encephalitis).

Family History.—The patient was a woman, aged 43, married, born in Italy. She had lived in the United States eighteen years and did housework. The history of her grandparents was negative. The father died of pneumonia at

35. Oppenheim, H.: Textbook of Nervous Diseases, translated by Bruce. London, 1911, p. 825.

73; the mother at 65 of an unknown cause. A paternal uncle died of heart trouble and his sister suddenly of unknown cause. A maternal brother killed himself while drunk (?). There are four siblings living and well, none dead. No mental, nervous or epileptoid disease was acknowledged.

Past History.—The woman had been taught in Italy to read and write, but little more. She was always a healthy child and was never in a hospital. She was married at 27 to a man of 46, and the union was happy and fruitful. There are nine children living and well; one died of acute indigestion. There were no miscarriages, no known mental defect in the children.

She never did other than housework. Her personality is described as being "quiet, not inclined toward recreation, but prefers to stay at home. Good-natured, and a good housekeeper. Normal interest in her (Catholic) religion." She was not alcoholic.

Present Illness.—Until September 10 the patient was considered well in every respect. On that date she went to bed with an acute attack of influenza. There is some question as to the duration of this disease. She is known to have been febrile for two succeeding days. She then tried to get up, but had "a spell" of an hour's duration during which she was tremulous, "nervous," suffered from palpitation, and had a fear of death. That night she was unable to sleep for fear, and this symptom persisted. She was taken to a general hospital and spent fifteen days there, during which time she was constantly agitated and querulous, complaining of multitudinous vague pains in stomach, head, etc. She went home and remained in bed for two weeks; then she was up and about for some six weeks more before being brought to this hospital. She was thought by her relatives to exaggerate trivial symptoms; she complained constantly, believing her digestion impeded, that she was hopelessly constipated, and that she could not possibly live. She made many pretended attempts at suicide and finally one confessed bona fide attempt.

There were no avowed hallucinations, no observed fainting attacks and no convulsions; and there was no delirium, no known amnesia, no fibrillation, etc. It was about ten weeks after the onset of the influenza that she was brought to this hospital.

On the day of admission, when she tried to cut her wrist vessels, "she was excited for the first time, and pushed her husband away and did not want to see any one." The admission notes at this hospital, condensed, read as follows:

"Patient is an agitated and apprehensive Italian woman who answers questions promptly and accurately. Since influenza in September . . . she has been depressed, afraid she would die, worried over her physical condition. 'I feel sick all time. I think I die. If I shut my eyes I see bad people with big eyes. If I go to sleep I wake up and worry and think never get well. It all is come for my stomach. If I eat I feel bad; if I don't eat my stomach empty, I feel better. My nerves are all excited and my nights are terrible.' She complains of palpitation, constipation, and insomnia in addition. No conduct disorder, and no frank evidence of depression."

The ward notes made thereafter state that no change in general demeanor occurred.

Mental Examination.—Appearance and behavior were as given above. Her orientation was precise. Her memory was not defective either for recent or remote events. She gave the full details of her family and past history, as

well as of the present illness in accordance with the outside history as related. In regard to *hallucinations*, she declared that when she closed her eyes at night she saw the faces of dead people and others. This frightened her more than ever, but the visions disappeared when she opened her eyes. The visions did not speak to her—there were no auditory hallucinations. *Ideas and Judgment*: Delusionary formation and lack of insight may be noted from the above account. Her *thought processes* showed no manifest abnormalities of attention, associations, or train of thought. Retardation was not present. "She speaks in a rather melancholic tone, whispering at times. *Emotionally*, she is more apprehensive than depressed, and greatly worried. At times she smiles pleasantly, but there is no schizothymia." Her *motor status* showed mild hypokinesia with no further conduct disorder.

Physical Examination entirely negative, except that the knee jerks were not obtained. Blood pressure, systolic 118, diastolic 70.

Laboratory Findings.—Urine, blood serum Wassermann test and spinal fluid examination, including Wassermann test, negative.

Course.—It is interesting to note that on the fifth day after admission the staff made the following vote on the diagnosis: Psychoneurosis, 2; manic-depressive psychosis, depressed phase, 1; undiagnosed psychosis, 4.

On the 19th, six days after admission, she was found to have a pulse of 140, a temperature of 104, and was apparently unconscious. In this condition she remained for over two days and died.

A necropsy was performed by the assistant pathologist to the Massachusetts Commission on Mental Disease, Dr. Myrtelle M. Canavan, and the brain in toto and in section was examined by the pathologist, Dr. E. E. Southard. The chief findings were: An extensive cerebral hemorrhage of large size with evidence of many petechial hemorrhages throughout the cortex, and bloody fluid in the third ventricle. There were numerous "flea-bitelike dots" of blood between which the tissue was softened and of a gray to grayish-red color, as described by Leichtenstern. In addition, there was chronic fibrous endocarditis of left auricle and of mitral and aortic valves, old pleuritis, purulent bronchitis, gallstones in duct, slight vascular nephritis, aortic sclerosis, petechial gastric hemorrhages, hemorrhagic uterine lining, acute leptomeningitis.

GROUP 6.—*Somatopsychoses*. Psychoses with somatic disease.

Delirium in all its polychromatic variety may be proposed as the representative symptom of this group. In so declaring we elude the pregnant question of just what delirium really is. In the paper previously mentioned²⁴ I have made a plea for the readoption of the simplest of classifications for delirium, namely, one based on the time of onset with respect to the fever, of which it may, indeed, be entirely independent. Thus all deliria associated with infectious diseases may be designated as either prefebrile (cum) febrile, or postfebrile, and in such a designation there is the added value of a prognostic index. The postfebrile forms are proverbially difficult of diagnosis and grave in prognosis. The fourth form, afebrile delirium, does not enter into the present discussion.

The fact that no form of mental disturbance is more familiar than what is termed "febrile delirium" is far from equivalent to saying that

it is the best understood. The descriptions of its essential nature are widely variant; even now there is no agreement on its pathognomonic signs or symptoms, and many—the writer included—are inclined toward agnosticism in point of pathognomonic indices, and to doubt if any such exist. Part of our difficulty herein may be referred to the psychologists, who have not yet decided just what we should believe as to the nature of consciousness; but perhaps an even greater blame falls on psychiatrists themselves for the confusion arising from the use of inaccurate, ambiguous and indefinite terminology and nomenclature.

Of the various forms of transient mental disturbance whose general character fitted the hazy outlines of the psychosis "delirium," certain types were particularly striking. The "muttering delirium" of historic fame, the "typhoidal delirium," the acutely maniacal forms of delirium with homicidal tendencies, types simulating Korsakoff's syndrome, delirium without disorientation, and divers others were encountered. Two types deserve special mention. In not a few cases there was a history of wandering about the neighborhood unable to find the way home, pounding on the doors of houses in alarm or terror, disappearing from work or home and later discovered by the police, amnesic and disoriented. These usually occurred during convalescence. An informal and unofficial report from the medical officers of a certain naval prison is to the effect that a number of the prisoners provisionally classed as "deserters" absconded during the convalescent period following influenza, and it was found that some of these, at least, were amnesic for events of the period and represented, in the minds of the naval medical officers, additional instances of postinfluenzal twilight states.

A second unusual form of delirium is what might be denoted delirium schizoprenoides, the schizophrenic elements dominating the picture to such an extent that the differentiation from dementia praecox in the cross section was quite impossible. This type seems to have been exceedingly frequent after the recent epidemic, as it is mentioned in practically all of the reports on mental sequelae of influenza which have appeared. (Burr,²⁷ Egbert Fell,³⁶ Harris³⁷ and others.) Because of the frequency of this type in our series, and the remarkable configuration of the clinical picture, it is this type of which I have chosen to give an illustration. The following may be considered a representative example of this type.

36. Fell, Egbert W.: Postinfluenzal Psychoses, *J. A. M. A.* **72**:1658 (June 7) 1919.

37. Harris, A. F.: Influenza as a Factor in Precipitating Latent Psychoses and Initiating Psychoses, with a Brief History of the Disease and Analysis of Cases, *Boston M. & S. J.* **180**:610 (May 29) 1919.

CASE G.—*Normality (?) + Influenza = Delirium*; first maniacal, then schizophrenic, then depressed, then stupid; finally disappearing.

Family History.—The patient was a woman, aged 34, a Nova Scotian, married and had always done housework.

The paternal grandfather died at 72 of pneumonia. He was a Nova Scotian fisherman. The paternal grandmother, a "kind, sociable, Christian woman," died at 93 of pneumonia. The maternal grandfather died at 72 of Bright's disease; the maternal grandmother, at 64 of a tuberculosis which she was known to have had many years. Her father was a carpenter 62 years old, a church deacon, said to have cancer of the lip. Her mother, the chief informant, seemed to be intelligent and well informed. She thinks she has had heart and kidney disease for some ten years. The uncles were chiefly sea-faring men; those who were not drowned are in general still alive and well. A maternal sibling died of tuberculosis of the brain; a paternal sibling of consumption. Aunts numerous, nothing of importance. Siblings: Brother, 37, well; the patient, 33; sister 31, with two healthy children; sister, 29, with three healthy children and one dead from tuberculosis; sister, 19, died of tuberculosis; sister died of pneumonia at 3; sister, 22, well. No known mental or nervous disease in any branch of the family.

Past History.—The patient was born on the Nova Scotian coast in 1886, and lived there until the age of 22, when she came to Boston and has lived here since. Educational: She attended the grammar and high schools from the age of 6 to 17, with a good record throughout. Habits: No alcohol nor tobacco; her meals were regular, and her exercise fairly regularly.

Personality: "Active, lively, talkative, high spirited, kind, practical, independent. Fond of music, is much interested in the Eastern Star fraternity. Member of Baptist church." Later we were told, however, that she had always been extremely suspicious. Marital: She had been married four years prior to her present illness. Her husband was a rather egocentric individual, "demanding more than he gave." The union was happy, however. A child, born less than a year after marriage, is well and strong, although he had influenza and pneumonia along with his parents. Medical: Except for the diseases of childhood, the patient had no serious nor protracted illnesses except typhoid fever, which she had at the age of 14.

Present Illness.—Husband and wife were taken ill with influenza about November 17. On November 24 the husband died. Even before his death, the patient began to show definite evidences of psychosis. "The room was filled with the Holy Ghost." "The spirit of God" told her he would not die. She kept saying, "Don't you feel the stillness?" and made the family pray with her constantly.

The day after her husband's death she was up and out of the house, seemed fatigued, talked less, and was less religious. The following day she spoke of having died, gone to heaven and returned; insisted that she was God, and wished to convert everyone. The following day she went with her sister to attend to some business. She seemed alternately dreamy, deluded, semi-conscious, and in a state of complete clarity. On the evening of the latter day she showed evidences of great fatigue, "continued to scream, laugh, sing hymns, and wanted others to do so." This continued for the next day or so. She then began to claim supernatural power, talked of striking people dead or blind, insisted that she was God, and her child the devil. Because of her threat to show what she would do to "the devil" she was brought to this hospital lest she injure her baby. She was brought by the police.

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Mental Examination.—On the day of admission the patient manifested great excitement. She was constantly talking, shouting, or singing. A sample of her discourse at this time follows:

"Now I wish you to know that at this present time I am God. I am God. I am God. You understand, I am God. God says, 'Now let us sit down and reason together. Laugh and the world laughs with you.' Weep, now Father, I don't want to do that. God so loved the world that he gave," etc. (Where does that come from?) "The Book of God, do you know that?" (Then sang one verse of The Light of the World Is Jesus.)

(Why do you assume the personality of God?) "Write down for me the word G-O-D, God. You are fully convinced that I can see who I am, are you? Before I get through with you I will convince you that you don't know who you are, but I know who I am. I am M-M-M, and God is speaking through me. No, I am a scientist, I am only a Christian girl. The Blessed Virgin is right there with God, yes, she talks to me too. I can see them both either in daylight or at night. I have a mission on earth, it is to bury my husband, N-N-N."

At this time she was correctly oriented as to time, place, and, nominally, for person. Her replies to questions, while frequently irrelevant and incoherent, indicated that her *memory* for recent events was "not appreciably impaired." Her *ideation* showed expansive, delusionary formation, centering about a transformation of personality. Her *insight* did not extend beyond declaring that she was "a religious fanatic." Her *thought processes* showed unstable attention, loose and incoherent associations, and an accelerated train of thought without true flight of ideas. Irrelevancy in her replies was less common than incoherency in the statements themselves. At times she became obscene and profane. Her *emotional tone* was one of excitement, expansiveness, agitation without apprehension or elation. Her *motor status* was that of hyperkinesia in rather narrow limits. She was exceedingly untidy and kept her room and her clothes soiled.

This description is representative for the first five days of her stay. Thereafter the picture changed, as will be detailed below. The record of the following conversation was made on the fourth day toward evening, when she was rather less disturbed than usual. Except for the queries indicated in the parentheses the matter was entirely spontaneous.

She is in a private room, constantly jabbering and shouting, running about the room, crouching in a corner, or gesticulating. A stench of fecal and other origin fills the room. Her gown is soiled with a menstrual discharge, on which fact she comments unabashed. When the examiner opens the door she forces her way past him and leaps on a bed in the corridor. Here she perches immovably, silent for a few minutes at a time, but bursting out at frequent intervals in a senseless and incoherent comment on some word or phrase caught from an adjacent patient who is in a pack and much disturbed. (What are you doing?) "Why, I am rowing a boat. I am on to Bermuda. What you writing? (Snatches notebook.) Why don't you go and get me a glass of water? There, there, that's what it is. I am telling you what the nurses are doing. I will now convince you that I am not blind. You know damn well that I am going to unbraid my hair. If I get there. . . . You know damn well that . . . here . . . there . . . (her loosened hair falls over the bed railing). This is the fountain of life. Don't you touch that or I'll drive you through the wall so quick you won't know whether you are a bird or a nurse! Why, I told them you got all those supreme beings and the birds

and. . . See! See! That's what they do! Isn't it terrible! (Jumps on bed and assumes threatening attitude, but is quieted by nurse.) Get out of my way every damn one of you! Don't you dare to take that out of my mother's lips tonight, and I think it's a damn shame that I am still without that glass of water. You damn nurse I'll slap your face in a minute! You'll find your birds on that boat Bermuda, and I'm still without the glass of water I want a drink, you damn fool. (Overhears the word "skeletons.") I'll tell you who will be skeletons around here; the doctors and nurses. I'll poison every one of you. And I'm still without that glass of water. P, I, S, S, there, I'll call you a damn sweet doctor." (Nurse walks by.) Nurse says, "Excuse me." Patient replies, "Oh, you damn fool! (mimics) 'Excuse me, excuse me, excuse me,' what's that for?" (Water is brought. She obeys orders to keep her hands down and drink from the cup held by the nurse. Stops between draughts to say—) "And I'm still sitting here trying to get a drink of ice cold water. (Spits on the floor.) Now you are fully convinced that I did not have that glass of ice-cold water. (Supper is brought. She asks for sugar, is told that there is none, says all right she will drink the tea without it. Examiner remarks that she is a "great girl.") "You bet I am. That's the trouble, it's mind over matter. (Bursts into tears.) I'm crying to think I was treated so for calling you the sweet-faced doctor." (She hears another patient singing, and herself breaks into song, but in another key and tune.)

Physical Examination.—At this time the physical examination was essentially negative; the urine negative; the spinal fluid negative; Wassermann reactions on blood serum and spinal fluid negative.

Course.—The excitement gave way to a phase of inert apathy. She wandered about the ward, quiet and well behaved. She obeyed orders, retained her clothes, associated with other patients, but was rather noncommunicative, played the victrola, and was rarely or never talkative after the tenth day. On the fourteenth day irrelevancy, incoherency, inadequacy characterized her replies. Thus:

(What does God say to you?) "What's wrong? Nothing's the matter."

(What is the trouble with that girl?) (indicating a patient.) "I can only answer for one."

(Are you clear in the head now?) No reply. She stares inquiringly; glances furtively about.

(How do you feel today?) "It was, yes sir. Better."

(Are you better?) (After a pause) "My breath is very offensive."

On the sixteenth day she was quite disturbed in the forenoon, but toward evening became more composed. Cried when her husband's death was mentioned. Answers still illogical, irrelevant, incoherent. No insight. She talked a little of having been "a religious fanatic," but the words seemed to have no real meaning for her.

On the eighteenth day she was interviewed in continuous baths and was quiet and accessible. (How long have you been here?) "You know me as well as I know myself."

(What is that in your hand?) "My handkerchief."

(Why have you wadded it all up so?) "Why mother gave it to me."

(Why are you in the bath?) "She said it was necessary" (pointing to the nurse.

(Are you suspicious, Lillian?) "Of you?"

(Of any one?) "No." (Pause, looks at nurse) "But I know she don't dare wink any more."

(What?) "I said she wouldn't pull my hair again."

(Did she do so once?) "Ask her. Let her speak for herself. 'Speak for yourself, John.'—Miles Standish."

During the next week the patient seemed more and more quasi-normal, the chief symptoms being restlessness and the occasional expression of paranoid ideas. Irrelevancy and incoherence in her replies disappeared almost completely. A certain tendency to evasiveness was noted. About this time she was asked to write her version of her trouble, and wrote the following account:

"I was suffering from severe headaches for over a week and had been without sleep for one week previous to being brought here. I was so disturbed in my mind about where my husband's soul would go that I prayed with my Maker and asked for His help in my afflictions. I felt my prayers were being answered and I felt the presence of God around me. All people would say was 'sleep.' I became somewhat of a religious fanatic and heard voices speaking inwardly. I quoted passages of scripture and the Twenty-Third Psalm ran through my mind. I talked to those around me and spoke of the teachings of learned men that I had heard preach from the Bible. One day, in taking my baths, I spoke of Billy Sunday and told one of the patients about it. I was somewhat emphatic in assuming so much Godlike spirit, but my nerves were somewhat unstrung, and while I still feel the need of prayer I am able to control my nerves and know that I have been helped in my treatment here. It all came from my mind being overtaxed by thinking so steadily. But thanks to all around I feel my feet are on terra firma once more and I realize how disturbed I was."

Condition on the Thirtieth Day.—The patient had been transferred to the quiet ward and was daily employed in ward work and occupational therapy. She seemed, superficially, quite normal, with partial insight as to her recent mental trouble. She recalled many of the incidents, but was unable to recall many others. She claimed to have regained entirely her old interests and affects and was complacently waiting to be discharged.

Certain residual symptoms remained, however. A nurse's note is quite expressive. "Patient depressed at times. Cries easily, but says she is strong enough to go home. Seems suspicious. Thinks conversation of others is directed toward her. Asks the same question several times."

Moreover, she seemed somewhat slow in grasping the situation. She doubted if she were really to go home as had been announced to her and cried at the realization that it was true. She lingered about the ward office as if fearing something might be said about her. Her psychometric rating, moreover, gave her a mental age of only 13—on Point scale; $13\frac{7}{12}$ on Stanford. She was discharged on the thirty-first day, virtually, although not technically, against advice, improved but not recovered. The diagnoses varied widely, ranging from cyclothymic psychosis to dementia praecox, according to the period of consideration. On the whole the case seems to be quite representative of one of the polychromatic forms of postfebrile (influenza) delirium.

Subsequent Report.—Two weeks after discharge the patient reported. At this time she betrayed a peculiar paranoid state difficult to define. The following conversation is illustrative of the irrelevance, inadequateness and suspicion which characterized her manner. She insisted that she was perfectly well (as did also her mother).

(We are in doubt as to how much can be remembered by delirious patients. Will you help us? How much do you remember?) "Well, I shouldn't remember everything, should I?"

(I don't know, do you?) "Well, it should be a little hazy, shouldn't it?"

(I don't know, is it?) "Well, to a certain extent."

(To what extent?) "Well, I don't suppose I remember everything. Can you remember everything that ever happened in your life?"

(We want you to come in and do another psychologic test.) "What for?"

(I think you could do one perfectly now. You didn't before.) "Yes, I did."

(No, it wasn't so good.) "Well, let some one else do it. You do it for me."

(Did you think you were God?) "I never said that."

(Yes, you did.) "Doctor, I never said that at all."

(You admit that you don't remember everything.) "Well, I didn't mean what you mean."

(You admit that you said you were God?) "I didn't."

(But we have it written down.) "Oh well, that's different."

(But you said it, you know.) "Well, I didn't mean what you mean. You can't read my mind and I can't read your mind. No one can know what I felt unless he has been at death's door. Have you ever been at the brink? That's where I was. I had only a flicker of life. I felt my pulse—it was very slow—about 40."

(Was it then that you heard and saw God?) "I said I was the armor of God . . . I had . . . that's what I said."

(What did you see?) "I saw everything."

(Spirits?) "Yes."

(What were the spirits like?) "Doctor, don't you have a conscience? Doesn't your conscience tell you things? That's what it was."

(Did you see angels?) "Yes. They weren't like you, though, Doctor, not like you, waving around in the air."

(What were they like?) "Doctor, you can't experience it unless you have been at death's door."

(I don't want to experience it. I want to hear about it.) (Reply was inadequate—details forgotten.)

The mother seems to be an intelligent woman who appreciates the evasiveness and paranoid trend of her daughter's replies, but says that she has been that way from childhood up. "As a child she was suspicious and I had to take care lest her feelings be hurt by trivial things that didn't concern her. If I spoke quietly to one of the other children, she would suspect that I was talking about her. She is absolutely the same now as she has always been. You have to know her to understand her ways. That is her, all over."

The further course of this case will be recorded in a later article of this series dealing with delirium and its relation to schizophrenia. For the present it serves admirably as one of those baffling cases, of which we had so many, wherein there was a constant battle between the protagonists of a diagnosis of schizophrenia *paranoides* and the advocates of the simple designation delirium (*schizophrenoides*).

GROUP 7.—*Geripsychoses*. Psychoses of senile and presenile periods

In a post graduate lecture in 1893 Gowers³⁸ mentions a case representative of many others wherein full mental vigor was replaced by utter dementia in a senescent individual. The influence of the acute

38. Gowers, W. R.: The Nervous Sequelae of Influenza, *Lancet* 2:1 and 73, 1893.

infections is vaguely enough understood at best, and our knowledge of the effect on the mental diseases of old age is the least favored in point of available material. The following example speaks for itself. The writer is quite conscious of the deficiencies in diagnostic data, but the known facts are strongly indicative on the basis of probabilities.

CASE H.—Senescence + Influenza = Senile Psychosis, Paranoid Form.

Family History.—A married man, aged 61, born in Maine in 1859, had a negative family history.

Past History.—He had had the usual children's diseases, but no severe illness nor convulsions. After a cursory education he entered the shoe business and remained at that work consistently the rest of his life. Habits: He had been a total abstainer from alcohol. Marital: Married for forty years to one wife who died of "creeping paralysis," and for two years to a wife who is living and well.

Present Illness.—The patient was a Christian Scientist whose orthodoxy prevented his consulting medical advice and likewise prevented our acquisition of a good account of his trouble. The following admission note gives all that was ascertained.

"Influenza began approximately October 19. Although not bedridden, he was feverish for several days. Mental symptoms appeared a few days afterward, about ten days before admission, rather insidiously. According to his wife and a friend, extreme talkativeness, restlessness, insomnia, and delusions of government inspectors pursuing his friend are the chief symptoms. In the admission office he was very indignant, having been brought by strategy, and insisted on having it explained why he was brought, what the disturbance was all about, etc. He talked at a great rate about his unblemished record, and made remarks and hints which referred to the delusions above mentioned, but denied them when flatly put to him. Absolutely no insight. He has a marked aversion to doctors and hospitals. Quite resistant on way to ward."

The physician who examined him on the ward noted: "He is a very talkative and extremely irritable man, who can give no reason for coming here, but demands that some explanation be given him. He refuses to admit anything, even that he has just had influenza. Very circumstantial and rambling in his conversation. He is apparently well oriented and his memory seems very good. No delusions or hallucinations are elicited. Very irritable. Radial arteries markedly palpable."

Mental Examination.—After the day of admission depression, humiliation, and chagrin replaced the irritability and disagreeableness. He was a rather pitiful sight, sitting about the ward in great dejection, and while superficially accessible would give no adequate expression of his ideation except that friends had wronged him. His thought processes showed marked circumstantiality, occasional irrelevance, and a distinct tendency to wander from the subject at hand.

Emotional Tone.—Depression, irritability and agitation were represented at different times. No considerable amnesia, disorientation or disturbance of motor status and no hallucinations were ascertained. No further data concerning the delusions were obtained than those mentioned in the admission note, save that he was fearful that something would happen to him here.

Physical Examination.—He was fairly well developed, but poorly nourished; his hearing was slightly impaired; he had a slight tremor of the tongue, normal reflexes, no teeth. His lungs showed fair resonance throughout, except for a slight dulness in the right base, numerous râles in the left back, and a reputed friction rub in the left axillary region. Heart: The apex was felt in the fifth space. The right border was 2.5 c.c. to the right of the midsternal line; the left border, 9 c.c. to the left. Aortic and pulmonary sounds were of good quality; no murmurs were heard. The pulse was equal, regular, and synchronous. Arteries (temporal and radial) were tortuous and sclerosed. There was a pulsation of the neck vessels. Numerous dilated capillaries were found on the legs. The blood pressure was from 150 to 76. The physical examination was otherwise negative.

Laboratory Findings.—These were entirely negative, including urine, spinal fluid, and blood Wassermann test.

Course.—A lumbar puncture was done on the sixth day. He was kept in bed twenty-four hours thereafter. On the eighth day he complained of a little headache and some nausea. He made no other complaints and went to bed as usual on the evening of that day. At 8:30 p. m. the nurse entered his room and spoke with him. He was quiet, and there was no complaint. He was seen at least twice during the night by attendants, and nothing unusual was noticed. At 6:30 a. m., November 9, as he had not arisen when the other patients arose, the nurse went to call him, entered his room, and found him dead.

The medical examiner was notified and inspected the body before its removal from the bed or the room. He expressed the opinion that death was due to cardiovascular disease and did not recommend a necropsy. Whether or not this diagnosis by inspection postmortem resulted in a correct physical diagnosis, the fact remains that the psychiatric diagnosis of psychosis with cerebral arteriosclerosis is not tenable. Neither the amnesia nor any evidences of focal or general cortical irritation impairment, or destruction, were present. On the other hand, the vague persecutory delusions, the total absence of insight, the irritable, depressed and agitated emotional tones, plus the history of insomnia and restlessness, together with the age of the man, make a diagnosis of senile psychosis of a paranoid form the most reasonable hypothesis.

GROUP 8.—*Schizophrenoses*. Dementia praecox

The conception of schizophrenia as delimited by Kraepelin³⁹ and elaborated by Bleuler,⁴⁰ was not in the psychiatric mind at the time of the last great influenza epidemic. Consequently, there is not in the literature any elaborate discussion of the rôle of influenza in the production of the picture of dementia praecox. And, as stated in the introduction, no other acute febrile disease occurs in sufficient ubiquity and sufficient numbers to make possible accurate comparative study of its psychic effects under uniform extraneous conditions.

That influenza may be followed by schizophrenia is mentioned directly in a few instances, and indirectly rather often. Thus, Sir William Gowers³⁸ wrote in 1893: "Just as the depression develops

39. Kraepelin, E.: *Psychiatrie*, Ed. 8.

40. Bleuler: In Aschaffenberg's *Handbuch*, Leipzig and Vienna, 1912.

into melancholia, so the delirium which occasionally attends the acute affections may have for its special sequel chronic delusional insanity (read dementia praecox) and, very rarely, acute mania." Again, without entering into a discussion of the proper distribution of the entities composing the heterogeneous, ill-defined and fortunately obsolescent syndrome "amentia," it will be recalled that Kraepelin pointed out that many cases so diagnosticated proved to be dementia praecox, and that Régis⁴¹ regarded a prolongation of "Confusion Mentale" (essentially the same concept) as practically identical with one of his two forms of dementia praecox (constitutional and incidental). And as "amentia" and "mental confusion," etc., are frequently mentioned as sequelae of influenza in the older literature (including Régis' textbook), one may presume that the occurrence of dementia praecox after influenza, although frequent, by a confusion of nomenclature escaped signalization.

The few authors who mention it specifically do so with an apologetic manner, generally ascribing its occurrence to a coincidence. Kim, Bonhoeffer and others mentioned above have referred to it, but generally add reassurance that definite stigmata of psychotic tendencies were previously manifest, or were apparent in the family history. Paton⁴² remarks the occasional precipitation of Schizophrenia by influenza, but ascribes to it only a minor rôle.

These writers were in general still strongly influenced by the functional conception of Schizophrenia. The great majority of them were primarily clinicians. And because of the greater availability of material, the protagonists of the organic basis have attacked the problem almost altogether from the pathologic side. In the specific instance of influenza, Gosline²² reported a series of necropsies with histologic brain findings, and pointed out the similarity of findings in a case of influenza with delirium, and cases of dementia praecox, drawing the obvious inference, "that . . . certain cases of dementia praecox are due to infectious or toxic processes."

Perhaps the organicists have overlooked what might seem to give considerable weight to their thesis, accruing from the clinical side, as observed in the recent epidemic, and in the present series. In the statistical study previously mentioned²⁴ I pointed out the surprisingly great numerical incidence of schizophrenia, and concluded that "an organic basis for some instances of the picture denoted dementia praecox is supported by" this and other facts of that series.

41. Régis: *Psychiatrie*, Paris, and, *Délire de la convalescence*, Ann. méd.-psych., Paris, 1883, p. 393.

42. Paton, S.: *Psychiatry*, Philadelphia, J. B. Lippincott Co., 1905.

I now propose to present typical instances of this phenomenon.

Because of the emphasis placed on the matter of predisposition by most writers, two types of cases are recorded. The first two with a history satisfactorily demonstrating a tendency toward the seclusive and egocentric; and the third one clearly without any such stigma. As cases similar to both of these types were, as previously reported, by all means the most common forms of mental disease, the choice is rather wide. Many of the cases, however, have minor complicating factors: the history of a recent miscarriage; an incomplete record of the past history; defective laboratory reports, etc. In general, the cases are quite representative and fairly complete. Two instances of the precipitation of second attacks of the cyclic form of schizophrenia occurred. These are not here illustrated.

CASE I.—Predisposition (egocentric temperament) + Influenza = Full blown Schizophrenia.

Family History.—An unmarried nurse, aged 35, was born in Massachusetts. Her grandparents were born in Ireland, of whom there is no history. Her parents also were born in Ireland. Her father died at 65 with pneumonia, the mother, at 67, of shock. There were paternal and maternal siblings, of whom there is no history. The patient's siblings: two sisters, living and well; one brother aged 38; a twin sister died of gallstones. A nephew died of tuberculosis. No history of mental or nervous diseases in the family.

Past History.—The patient was born in a Massachusetts village. She graduated from grammar school. Not much is known of her childhood. At 26 she graduated from a nurse's training course and since that time has been a visiting nurse for the Boston Consumptive Hospital. *Medical:* The patient suffered a Colles' fracture five years previously and was described as having had "neuritis" for a period of seven months three years ago, which was connected in some way with her diet. "A stomach trouble" which was suspected by her friends to have been neurotic, had been complained of for many years. *Habits:* She took no alcohol whatever. She paid much attention to treating herself and lived on a special diet because of this "stomach trouble." Her work gave her a reasonable amount of exercise.

Personality.—The patient is described by the superintendent of nurses, who had known her for eight years, and who seemed to be an intelligent, reliable and observing person.

"She talks continually about herself. She has always been egotistical, selfish, self-satisfied. She is not popular, but she continually talks about herself and she was always looking for sympathy. No matter what subject is brought up she will invariably bring it back to her own illnesses. She has a few friends, but is definitely seclusive and does not go about with them much. She is loud and noisy in her actions, speech and laughter, and her laugh is empty, forced and hysterical. She poses as younger than she actually is and never realizes her age. She is either way up or way down. She is stingy with money, except for herself, and spends much on dress. She is a devout Roman Catholic."

Present Illness.—She contracted influenza on September 28. She had a high fever which disappeared on the fifth day. It is thought to have been as

high as 104 F. and known to have been 102, but she was at no time delirious. On the third day she seemed quite hilarious, saying that she had the influenza and might as well make the best of it. The next few days "she began to worry about her treatment as usual."

By the time her temperature had returned to normal, she was distinctly psychotic. She is thus described: "First she would be rigid and staring and would not speak. She would relax if grasped by the arm. At other times she would talk a blue streak. This was not irrelevant or incoherent, and she could tell the doctor accurately what happened during the previous twenty-four hours. She paid little attention to people or to things happening in the room."

On the ninth day she thought she was going to die and was anointed by a priest. She insisted that she still had influenza and said that she had been accused and told people not to come near her on that account. She slept little or none. At 3 a. m. on the tenth day she became violent, disordered her room and hurled objects at any one entering. She finally quieted down, but became excited and disturbed again the following day. Then she was seen by a reliable physician, who examined her carefully and concluded that, in spite of very active reflexes, she did not have meningitis which he had suspected because of an apparent stiffness of the neck. She was deaf for one day. She continued to show major and minor conduct disorder, but without any loss of memory or orientation, and without hallucinations, suicidal threats or homicidal ideas. Delusions were very vague. She would make such incoherent remarks as, "I have picked out a rat hole," and "I am a slacker and I am not going to die." She was sent to this hospital on the eleventh day.

Mental Examination.—The patient was never fully accessible. For the first few days she answered questions, but with varying degrees of relevancy. Sometimes she would make sensible and accurate replies to question, but at other times she would refuse to answer or would reply with irrelevance, nonsense or inadequacy. After the first few days she became almost completely inaccessible. She mumbled frequently to herself about being cursed, going to hell, etc. She whispered to herself, attitudinized, gesticulated dumbly, answered hallucinatory voices, occasionally jumped impulsively from her bed and walked to one corner of the room, and returned to bed without a word, repeating the process for each corner of the room. She admitted hallucinations of God's voice, and those of various persons.

Subsequently, she lay almost motionless in bed, completely inaccessible, almost completely mute. Her face wore at times a pained, worried expression, but in general it was masklike and utterly expressionless. She showed negativism, catalepsy, and on a few occasions a transient *flexibilitas cerea*. She occasionally answered questions to the extent of complaining that her mind was being influenced, that people will not let her talk, that she was dead, etc.

One or two days during the second month she would answer a few questions relevantly and correctly, but with an utter indifference and with a far-away stare and absorbed manner which is difficult to describe, but which is perfectly familiar to any one with psychiatric experience.

At the present moment, three months from date of admission, she remains precisely the same. Occasionally she bursts into a mumbled jargon of incoherences accompanied by a silly, meaningless smile; at other times she renders a loud, wild shriek or moan without any apparent cause whatever. But in the main she lies stolidly in bed, making no motion, speaking no word. Catheterization and tube feeding have occasionally been necessary.

Physical examination and laboratory findings were negative.

Diagnosis.—Catatonic schizophrenia (dementia praecox).

Another case further illustrating the same paradigm, but in the male sex, follows in abridged form.

CASE J.—*Predisposition (seclusive temperament) + Influenza = Schizophrenia.*

Family History.—The patient was a soldier, aged 27, single, and gave a negative family history.

Past History.—He was born in Vermont in 1891. His past history is not important. In temperament and disposition he is described by his brother as having been always of a quiet, seclusive disposition, never mixing with others, but preferring to remain alone. He was considered normal mentally by his employers, friends, and family, and he was not regarded as eccentric.

Present Illness.—He developed influenza and pneumonia at Camp Devens where he was a private in the infantry. He was delirious and did not regain his mental faculties. He knew his mother and father except on their last visit to him, at which time he had evidently recovered from everything except a mental disorder. His brother saw him two weeks ago and was recognized by him then. An interested friend writes: "I am informed that when he took sick he was out doing trench work, was missed at roll-call, but was not found until next morning, lying in the trench where he had been working after having lain out all night in a cold rain."

The military authorities sent only the information that subsequent to the influenza "he has been in a catatonic stupor; eats little; unclean in his habits; absolutely mute and unresponsive to external stimuli." He had been in the psychopathic ward of the base hospital for two months.

Mental Examination.—He was never accessible. He lay passively and apathetically in bed, responding to no questions by look or word. He obeyed simple orders, however, and cooperated in a fair way in the neurological examination. He made very mildly resistive maneuvers at times. After the first few days he was up and about the ward and was seen to look through magazines. He continued to show hypobulia and complete apathy. When offered a hand, he presented his own, and shook hands listlessly. He could not be urged, persuaded or forced to speak, smile, laugh, or cry. His facial expression remained fixed and impassive and his thought processes showed complete blocking. He seemed at no time unaware of his environment; his reception of external stimuli was not interfered with. Thus, when told to indicate by signs his interpretation of some object, he did so slowly and disinterestedly, but quite correctly.

Physical Examination.—This was negative, as also the laboratory findings, including spinal fluid, and Wassermann tests were entirely negative.

CASE K.—*Normality (!) + Influenza = Schizophrenia.*

Family History.—A single woman, aged 20, was a bookkeeper by occupation and was born in Massachusetts. Her grandparents' history was negative. Parents: Father, 42, salesman; temperate user of alcohol; well. Mother, 41, always strong and well. Although described as being of a nervous temperament she was cheerful and not seclusive or sensitive. She had a premature menopause at 28, at which time she had "hysterical attacks." In these attacks she became excited, exhibited mild conduct disorder, and sometimes fell to the floor

with flushed face and some frothing, "but never lost consciousness, bit her tongue, injured herself" or passed urine or feces. Since the abatement of attacks she has grown entirely strong and much less "nervous" and "now has good self-control," and a cheerful, even temperament.

Past History.—The patient was born in Chelsea, in 1898; normal but somewhat difficult delivery. She was developed normally, physically and mentally. Puberty occurred at 13 years; no disturbances. Education: She started to school at 5, quit at 16, two years of high school, one double promotion at 11 or 12. Never held back. Did well; mathematics difficult. Very quick in languages. Never held back or kept out. Economic: From 16 to 18 she was home with her mother. At 18, she became a bookkeeper at \$12-a week. Has worked steadily with little vacation for the same people; eight-hour day but little responsibility until last three months. No court record or marital history.

Personality.—Lively, talkative, systematic, practical, level headed, social "but does not care particularly for social functions"; sympathetic, generous, sociable at home and domestic. Goes to theater with her mother. Plays piano. Has many friends. Pleasant, even disposition. Not easily influenced." (Given by father and mother who seemed to be intelligent and honest informants.) Religion: Protestant, little interest. Habits: No alcohol. Meals, wholesome and regular. Does not care for men. Thrifty, and a good housekeeper. Medical: Whooping cough at 6 months. At 1 year, measles (slight). At 18 months, tonsillitis. Has never been ill or had a physician since. No nervous attacks.

Present Illness.—"Patient is very ambitious and since the head of her department went into the army in June, she has had great responsibility. Has had charge of payroll of 150 people, directed the bookkeeping and practically the entire office. Worked very hard and loved it."

September 28 patient went to bed with influenza. She was febrile for ten days, but not delirious. On the twelfth day a neighbor who is described as being a "religious fanatic" called on the patient and the parents ascribe to this interview a psychogenetic influence which it probably does not deserve. However, that evening the patient was very depressed, wept, and said she knew she was going to die. As a matter of fact she was much better. Three days later the family moved, although the patient was still bedridden. She continued to improve. However, four days later (nineteen days after the onset of the influenza) she became mute.

"She has not said a word to her father since then, although she had what seemed to be normal periods. Her mother said that she appeared more herself in the morning when she had had a good night than later in the day when she had grown tired. A menstrual period due on the nineteenth was missed for the first time in her life. She grew steadily worse; more quiet, apathetic, and showing minor conduct disorders. On five occasions she wanted to undress at improper times and places. Twice without apparent reason she went out in her nightdress. Occasionally she emitted bursts of laughter or weeping without adequate cause." She was brought to the hospital on November 6, still completely mute.

Mental Examination.—A fair, comely girl, rather slight, though well nourished with a face of intelligence and culture, but entirely devoid of expression. She practically never spoke. All queries were met only with a Mona Lisa smile, and no indication that they were heard. Once after persistent questioning she showed some annoyance and ejaculated, "Damn!" at one other time she said two or three incoherent words. Occasionally smiled. Otherwise there was no

response whatever at any time. A nurse's note has it that she occasionally spoke to them and that she sang a few times while in the continuous baths.

Motor Status.—She was quite resistive to any examination, and gynecological was impossible. She ate and slept fairly well, but showed a distant aboulia, sitting about quietly without any show of interest. Frequently she would suddenly jump from her chair, run toward one of the physicians on the ward, and just before reaching him stop short, giggle in a wild and strange manner, and walk quickly back to her seat.

Emotional Tone.—She showed complete apathy. Occasionally there was some irritability; generally she wore a pleased expression and in a few days she seemed to be mildly interested in her environment, but in general she was quite indifferent.

Physical Examination.—This was entirely negative except for hyperactive knee jerks.

Laboratory Findings.—These were entirely negative, including spinal fluid and Wassermann.

Diagnosis.—She was committed after two weeks with a diagnosis of schizophrenia.

GROUP 9.—*Cyclothymoses.* Manic-depressive psychoses

Gowers' dictum on the frequency of "melancholia" and the rarity of "mania" after influenza has already been mentioned, and as it is a representative conclusion of the psychiatrists who wrote concerning the 1890 epidemic, no others will be quoted here. It is an amusing commentary on the older diagnoses, that in a recent examination of the records of a state hospital for the years succeeding 1890, the diagnosis "melancholia" was almost universal on all cases suspected of having had grippe. This in spite of quite good clinical descriptions which clearly made out cases to be schizophrenic, maniacal, delirious, etc. I have previously pointed out²⁴ that in psychiatric subjects depression is distinctly one of the less common symptoms. Manic-depressive psychosis appears, however, in episodes usually of the manic form, with considerable frequency in our series, in contradistinction to the reports of Gowers and others, and possibly due to more than mere differences in nomenclature and nosology.

Under this group three cases are here reported. In the cyclothymoses (or as I would prefer to denote them, the cyclopsychoses) there are, as with the preceding group, the individuals who have manifested a cyclothymic tendency either by attacks or by disposition, and those in whom no such history appeared. On this basis the cases given below were selected, to illustrate

CASE L.—Cyclothymic psychosis, manic phase, previous attack.

CASE M.—Cyclothymic psychosis, manic phase, no previous attack.

CASE N.—Cyclothymic psychosis, depressed phase, no previous attack.

(An instance of the occurrence of the depressed phase in second or third attacks could be added, but the analogy to Case L is so obvious that it is omitted to save space. Only a few such occurred.)

CASE L.—*Cyclothymic Tendency* (Previous attack of psychotic depression) + *Influenza* = *Hypomania* (Cyclothymic psychosis, manic phase).

Family History.—The patient was a man, aged 22, Jew, born in the United States of Russian parents. His paternal grandparents both died of pneumonia; maternal grandparents "of old age." Paternal uncles living, three; dead by trauma, one. Maternal side: one uncle insane, "was quiet and would not speak," died at a Massachusetts state hospital at 38 after three years' residence. Father, aged 50, living and well. Mother, aged 45, said to have diabetes.

Only the one instance of mental disease in the family history.

Past History.—Born in Massachusetts; negative history of birth, infancy and childhood, except for enuresis until 14 years old. Attended school from 4 to 20, taking part of the high school work, including the commercial course. Was obliged to repeat the first, second and sixth grades. Deportment always good.

Occupation.—Left high school to go to work as a clerk in his father's store. Joined the army August, 1918.

Medical.—Diphtheria and smallpox in youth. Gonorrheal infection at 20. Herniotomy a short time prior to this.

Habits.—Total abstainer from alcohol. Masturbation from 6 to 12, and sexual promiscuity from 7 till the present time. "Cabarets and vaudeville have been his chief forms of amusement."

Disposition.—Very happy, sunny, constantly active, with a fondness for work and disinclination for reading and religion. Occasional spells of depression.

Previous Psychotic Episode.—Two years previously he had had a distinct phase of depression lasting two months. He did not receive hospital care.

Present Illness.—While at Camp Devens, a private in the infantry, he contracted influenza of moderate severity and duration. During convalescence he first developed apparently a short depressed phase, succeeded by increased activity and elation which necessitated his transfer to Boston. (Details on military cases were never satisfactorily obtained.)

Mental Examination.—He was an alert accessible, and loquacious young Jew of rather high empathic index; he talked very freely, with circumstantiality, frequent abrupt alterations of subject matter and manifestation of a crowd of ideas. No disturbance of orientation, memory, or thought content was demonstrated. He had a few fleeting phantasmagoric ideas which could scarcely be called either delusions or hallucinations; e. g., he said it "seemed as if all the girls with whom he had ever had sexual relations were in the palm of his hand." Gross conduct disorder was never observed; he was rather hyperactive, but easily controlled, and highly appreciative of efforts in his behalf, such as the prolonged baths, which he enjoyed immensely. Emotionally, there was a typical elation of mild degree.

Psychologic test rating: mental age, 16.

Physical Examination.—Well developed and well nourished; hyperactive reflexes, pseudoclonuses of ankles. Blood pressure: systolic 120, diastolic 70. Otherwise entirely negative.

Laboratory Findings.—Urine, blood cytology and serology, and spinal fluid examination complete, all negative.

Diagnosis.—Hypomania.

CASE M.—*Normality (?) + Influenza = Cyclothymic Psychosis, Manic Phase.*

Family History.—A boy, aged 17, was a student and was born in Massachusetts. His family history was negative in all respects, including his grandparents. There were four siblings, all living and well, and no miscarriages.

Past History.—This was carefully obtained in the outpatient department of this hospital when the patient was about 12. It was in all respects entirely negative. His birth, infancy and childhood were entirely normal. He talked at 8 months, walked at a year, attended kindergarten, grade, and high schools and did good work. He was studious but not seclusive and evidently had a rather superior personality. He was athletic and interested in tennis, swimming, etc.

From the age of 7 until the age of 13 he was troubled by stammering. He attended a class for speech impediment in the outpatient department of this hospital, and was very much improved after a year or so that he discontinued his attendance. Except for this his medical and mental past history is entirely negative.

Present Illness.—He was working hard at various school activities in a boys' academy, having many duties in addition to his curricular work. Besides a scholarship, he had earned some \$300 at outside activities, and had kept in excellent health until October 1. At that date he contracted influenza and was very ill, running a temperature of 105.5 F. for three days. He was able to be out by the sixth day, but a cough and much restlessness continued. He played tennis, went automobiling and took a short vacation, but continued to show a distinct hyperlogia, making extensive plans for the immediate and distant future. On the twelfth day this became very noticeable. He "talked rapidly from one subject to another," spoke of being nervous and wondered if he wouldn't go crazy. He was given morphin by a private physician, but without apparently much effect. His hyperactivity continued, and he was brought to this hospital at 1 a. m., October 13.

Mental Examination.—"The patient is rather tall, slender, attractive youth of 17 who is hyperkinetic, talkative, and elated. He confers various military ranks on the physicians in the hospital, elects the nurses to the Red Cross service, and announces large salaries for the attendants, etc."

"I am absolutely perfect. Have a cigaret? Here are two strings which they gave me for a test. Hello there, Major. We are all going to be in uniform before night. How old are you? I am 17 years and 9 months today and in 3 months I will receive a commission. . . . Girls? Yes, girls by the thousand. Girls from Wellesley, girls from Dartmouth . . . no, there are no girls from Dartmouth . . . girls from Smith, girls, girls. We'll put this thing across, and have all those beds put in. Can you see it? Will you help it? Never mind, not necessary." (Whistles!)

The patient showed hyperlogia and hyperactivity as were indicated from the day of admission until discharge on the tenth day. He amused himself and his audience greatly, performing antics unnumbered, and of infinite variety, particularly when in the prolonged baths, and particularly when observed. *Emotional tone* was that of elation, exaltation, euphoria. His *thought processes* showed typical flight of ideas, with distractibility, play on words, etc. His *associations* were easily followed, however, for the most part. *Delusions* were

fleeting, and of the expansive and grandiose type, never paranoid, somatic or referred. There were no *hallucinations*. The *conduct* was as described above; hyperkinesis over a wide range, but usually readily controlled. He was obedient and not destructive.

Diagnosis.—Cyclothymic psychosis, manic phase. Committed.

CASE N.—*Normality (?) + Influenza = Cyclothymic Psychosis, depressed phase.*

Family History.—This patient was a man, aged 24, single, a chauffeur by occupation and was born in Boston. His grandparents were all born in Ireland, and died there at well advanced ages. His father was born in Ireland, a citizen of United States, a freight-handler, aged 51, without abnormalities of mental life. His mother was born in Ireland, aged 48, a charwoman, quite healthy. Possibly both mother and father are at least mildly alcoholic. Siblings: of parents, no social or mental pathology. Of patient, 8, 3 dying in infancy of enteric diseases, the others living and well. No history of mental pathology anywhere in the family. Excessive alcoholism also denied.

Past History.—The patient was born in Boston in 1894, and according to the history furnished by mother was evidently a normal infant and child. He left school at the seventh grade to work, was 14 and was considered a good student; had not been held back.

His *economic* history is rather varied, but not important. For some years he had worked as chauffeur, earning from \$20 to \$25 weekly.

Personality.—"He has a happy, sociable disposition. Has been very good to his mother, giving her most of his wages. Is fond of motion pictures, Chinatown suppers, and social parties. Is much liked, and is not suspicious." Catholic. Habits: Occasionally drank beer; "never seen or suspected of being drunk." Medical: Scarlet fever at 4, very severe and protracted. Diphtheria at 7. Since then never sick, until present illness.

Present Illness.—Ten weeks prior to admission he is said to have had influenza. "He was very ill. Temperature could not be taken because his teeth chattered so. He was in bed 7 or 8 days. He had night sweats (thereafter) for about a week."

He returned to work after two weeks, "but looked pale and weak but felt pretty well." He remained at work a week and "had a relapse." "He had a chill, sweats," and probably fever. He was confined this time for nine days, but was not in bed throughout this time.

Thereafter he felt quite incapable of returning to work. He "thought that he was dying," that "some one had given him the disease a second time," that "he couldn't seem to take hold and do his work." This continued for two weeks.

Two days before admission he called to his mother, asking what it was that was in bed with him, that it had frightened him. He was not suicidal, however, nor did he show conduct disorder nor react more definitely to hallucinations.

Mental Examination (abridged).—He wore constantly a pained, doleful, anxious expression. He was accessible, and tried to cooperate, but was not very successful because of his aboulia, retardation, and a suggestion of impoverished, intellectual processes. *Orientation* precise in all spheres. *Memory* general, rather faulty, perhaps due in part to abstraction. No *hallucinations*. He had an imperfectly formulated *delusion* that he had recently contracted venereal disease. No others. There was partial *insight*. . . . "Sometimes I don't seem right." His *thought processes* showed a conspicuous retardation, with

rather weak associations and an attention difficult to secure or retain. His *motor status* was distinctly that of hypokinesia and abulia. He sat about the wards all day, with head hung, and without demonstrating interest in anything. When obliged to move he did so with slow, irresolute movements, and with an air of lugubrious torpor. His *emotional tone* was incontrovertibly that of depression. He was "blue," "lonely," "worried," "down-hearted." He was never seen to weep, or to smile; he commented once that "some days things seem dark, and other days brighter."

Physical Examination.—This was entirely negative except for exaggerated reflexes and a tremor of the hands.

Laboratory Examinations.—These were negative, including spinal fluid and blood Wassermann tests.

Results.—His condition remained unchanged. He was removed against advice on the ninth day.

Diagnosis.—Depressed phase of cyclothymic psychosis (manic depressive, depressed).

GROUP 10.—*Psychoneuroses*

There is a book (4 vo) written in 1890, and now little read, entitled "De l'hystérie consécutive à la grippe" (Le Joubioux⁴³). The phenomenon referred to is striking and frequent enough to have attracted attention after each of the later epidemics of influenza. Grasset, Krannhals, van Deventer, Worms, Josserand, Huchard and many others have contributed reports and discussions, the consensus of which is well put by Leichtenstern.¹⁸ "Hysteria and neurasthenia not infrequently arise from influenza or are enormously exaggerated by it. All forms of hysteria have been observed. . . ."

He then asks the important question, "Is a neuropathic predisposition the basis of hysteria and neurasthenia when following influenza?" and answers "probably . . . in the affirmative, in most cases. On the other hand . . . severe postinfluenzal hysteria has frequently been observed . . . (where) no hereditary or acquired neuropathic tendency could be found." In this he simply speaks for a number of observers, some of which he quotes.

To circumvent the question, I present herewith two cases, one having a definitely manifested predisposition to psychoneurotic episodes, the other quite without any such tendency.

CASE O.—*Psychoneurotic Tendency* (manifested by a previous "hysterical attack") + *Influenza* = *Psychasthenia*.

Family History.—The patient was a woman, aged 34, born in Cambridge. Her paternal grandparents were Irish, both lived to be over 90; always healthy and temperate. Her maternal grandparents were Irish and English; both died "of old age" in the eighties; also healthy and temperate. Her father was born in Ireland, and came to United States when 12 years old. He died at 71 of

43. Le Joubioux: Thèse de Paris, 1890.

"cancer of the intestines." He was a railroad night-watchman, temperate and healthy. Her mother was born in Ireland and is living and well, aged 73. Paternal siblings, 9; maternal siblings, 8; all are living and well. There had been one miscarriage, at 6 months, during typhoid fever.

Past History.—The patient was born in Cambridge, Oct. 29, 1884. It was a normal delivery, a healthy baby, and she passed through a normal childhood. Puberty occurred at 14 or 15 without disturbance or illness.

Education: She entered the kindergarten at 3. Went through grade schools and finished the second year of high school at age of 15. Considered "very bright" and had "double promotion" twice.

Economic history: On leaving school worked two years as a night telephone operator. Mother objected, and so she remained at home for a year or more. From 21 to 30 she was employed as a saleslady by one firm, at \$10 a week. Has not worked steadily since (vide infra).

Not married. No court record. Habits normal. Religion: Catholic, devoutly held. Medical history negative except for the surgical removal of a small cyst and the appendix in July, 1917.

Personality described as "sympathetic, very sensitive, but not suspicious or jealous. Unselfish, very sociable, never irritable or critical. Fond of music and singing. (The mother, who gave the account, adds that "for a year I have not let her play because it seems to make her nervous.") It is thought that she was influenced rather with difficulty.

Previous Episodes.—The patient is well known to the outpatient department of this hospital. Four years previously she suddenly became "hysterical" at the sight of a brother dying with tuberculosis. She failed to recognize him [sic], cried "uncontrollably," and remained depressed and lugubrious. Her own account is identical, in that she explains that she was so "shocked" by her brother's appearance and so appalled at her helplessness in the situation that she had "an hysterical collapse" and was led from the room "screaming and crying." She was not able to work thereafter with any degree of proficiency. Although better at times, and particularly when under the surveillance of the social service department, she was nevertheless prone to "attacks" of profound despondency and weeping, once a month or less frequently. The social service worker finally established her at knitting on a commercial basis in her own home and she was diligent and efficient thus employed, knitting over fifty sweaters in the past year, and coincidentally feeling better than previously. She was consistently and persistently followed by the outpatient department and a voluminous literature has accumulated in her folder. The diagnosis there made was psychasthenia. She was considered much improved and had not been seen for nearly a year by the physicians.

Present Illness.—On October 10 she contracted influenza. "She had a temperature of 102 and 103 F. for the first few days. The fever did not pass for ten days; she was in bed about three weeks. For about two days, at the beginning, she was out of her head." Thereafter she seemed much the same for three weeks. On November 21 she complained of severe headache "and mumbled things to herself. She seemed much distressed and afraid of going crazy." She was brought on the following day to this hospital.

Mental Examination.—A young woman of rather large frame, well nourished, attentive, accessible, and cooperative, but very much self-centered. Consciousness entirely clear at all times, speech normal, orientation precise, memory free from defect. She gave a rather more detailed account of her past than that

furnished by the outside informant, but the main facts coincided throughout. She denied any stigmata of masked epilepsy (enuresis, somnambulism, etc.). Obsessive, obscene thoughts began to trouble her shortly after the attack of four years ago. She said they did not seem to originate from any particular cause of experience and they persisted for a year and a half. After the consultations in the outpatient department this improved and she was comparatively free from these thoughts.

Her account of her present illness is as follows: For some time she has been fairly comfortable mentally and physically and busily engaged in knitting sweaters at home, proceeds from the sale of which afforded her some little income. After a prolonged attack of influenza she began again to be troubled by obscene thoughts "much more than ever before." She told no one about them for two weeks, but they became so distressing that she could endure it no longer and following an expression of her feelings she was brought to this hospital. These thoughts are obscene and sexual suggestions and phantasies which "possess her whenever she is approached by any person." They come in spite of her dislike for them and her avowed efforts to keep them from her mind. They seem to have no particular reference to certain types of persons, they were not accompanied by hallucinations. "Something seems to grip my brain." After the thought comes the regret and chagrin that she should have been guilty of such thoughts. They seem to come in distinct showers; thus "today had none until about 3 p. m., when on roof garden" and "frequently has them after retiring at night." Latterly the obscenities became related to religious matters, but she was unable to explain this adequately.

In addition she had some symptoms of the "*folie de doute*." Thus for example she said "Sometimes I seemed compelled to repeat sentences, things I hear. If some one says breakfast is ready I feel as if I must keep repeating to myself, 'breakfast is ready, breakfast is ready.'"

She was precisely oriented, showed no memory defect, no hallucinations or illusions, no delusions, good insight and judgment, a rather variable emotional tone swinging from depression when introspective to pleasant, cheerfulness when distracted, normal thought processes and motor status. She was distinctly suggestible and was considerably improved by unsystematic suggestive therapeutics.

Physical Examination.—This proved to be essentially normal in all respects. Reflexes were slightly hyperactive; blood pressure was systolic 140, diastolic 80.

Laboratory Findings.—These were negative as to urine, blood serum, spinal fluid and vaginal smear.

The patient was discharged considerably improved after two weeks, with the diagnosis of psychasthenia.

CASE P.—*Normality + Influenza = Hysteria.*

Family History.—A girl, aged 15, was a student, and was born in Massachusetts. Her father was living and well, aged 52, not alcoholic. Her mother, aged 49, was always very healthy except for a gradual increasing deafness for the past fifteen years. "She has always been rather nervous and high strung and recently more irritable than usual." The patient stated that the entire family were "rather nervous and easily excited," but there was no history of nervous, mental, or epileptic disease in the family. One sister, aged 18, is healthy. Two siblings died in infancy of unknown causes.

Past History.—The patient was born in a suburb of Boston in 1903. She was rather weak and "sickly" until the age of 5, but since that age had enjoyed

unusually good health. Aside from the usual diseases of childhood and an occasional "cold" she had never been ill. There was no history of convulsions or other stigmata of epilepsy.

Educational: She started going to school at the age of 6 and was at the time of examination in the third year of high school. School work was always done very well and with genuine interest. Although she found her studies easy she was rather too assiduous and studied late into the night.

Personality: She seems to have been always a cheerful, happy individual with many friends and with one particular chum with whom she had associated closely for three years. Although her interests seemed to be confined chiefly to her school work she was definitely social, enjoying a crowd and disliking very much to be alone.

Present Illness.—The patient herself gives a very good account of her present illness, and it is here reproduced with minor modifications. An attack of influenza began November 2. She was in bed for about a week. She had a temperature as high as 103 F. There was apparently a good recovery after this, but about two weeks ago, since she started back to school, she noticed that she tired very easily, and felt she was overworking. She admitted that she had been studying more than usual in order to make up for the time she had missed.

The day before coming to the hospital she felt particularly tired. She had been studying late the night before, preparing for an examination, and felt so tired the next morning that she did not eat any breakfast, and also missed her lunch while at school. That afternoon she went to her friend's house, and while there became even more worn out. She said she did nothing out of the ordinary that afternoon, but after she had been there awhile she became rather silly. She felt that she was rather excited, and would laugh without provocation, and would say foolish things. Soon, however, she quieted down, and while sitting and talking realized suddenly that her left leg felt as if it had gone to sleep. She could not feel anything at all—felt as if it were gone. Tried to stand up but it collapsed under her, and she saved herself from falling by grasping a chair. This frightened her and she thought that perhaps she was going to be paralyzed. Then the other leg began to feel the same way, but not so markedly. The doctor was called soon after this, and he said it was nothing but nerves, and that she would be all right. This comforted her and although she felt too weak to go to her home she spent a restful night at the house of her friend. The next morning she was feeling entirely all right except that her legs were sore, and she had a rather severe pain in her back. She endeavored to get dressed so that she could go to her own home, but the pain in her back and legs would increase on any attempt of movement. That afternoon she thinks she must have become delirious because there are periods concerning which she can remember nothing. She would come out of these confused periods, and have no complaint except the soreness in her back, but soon she would drift away into unconsciousness. Once or twice while she was unconscious she felt that she was blind. She could see nothing, but would talk to the people around her and ask them why the room was so dark. They told her afterward that the light was on and that they could see perfectly clearly. These periods of blindness lasted only a few minutes. She knows nothing about what she said or did during the periods when delirious, although since she has been told about it by her family. She was afraid this time that they were going to take her away, send her to some hospital, and she would not be able to see her friends. She thinks she probably talked about this during the delirious periods.

She still was rather confused and uncertain when she came to this hospital, but the following morning felt entirely normal and has been that way ever since. She tells also of "an occasional lump in her throat, causing her to nearly choke." When asked about her girl friend, she admitted that she cared for her very much, but denied anything of a homosexual nature in their relations. Nothing of this kind could be elicited in any way. She admitted she had done a great deal to further the match between her friend and her cousin who is in France. She even wrote letters to her friend and signed them with the name of the man who is in France, but said she told her friend about it a month or so afterward. There had been no deception for at least three months on her part. She did not know whether the girl had been deceiving her or not.

Mental Examination.—Orientation precise, memory absolutely unimpaired except for certain recent events as detailed above. No delusions, no hallucinations, good insight. Thought processes show excellent attention, quick and coherent associations, normal train of thought, speech free from fault. Emotionally cheerful and pleasant. Conduct above reproach.

Physical Examination.—Entirely negative in every respect. The same is true of the laboratory examinations which included spinal fluid, blood serum, urine and vaginal smear.

Psychologic Examination.—The patient graded regularly at the mental age of 18 plus. Her performance on the construction puzzles was fairly good and on the memory tests good. In the suggestibility tests she accepted ten of ten suggestions. Patient cooperated well.

She showed no further evidences of mental disease during her stay here. She gave the impression of being "high strung" and "of an hysterical type" to various physicians who examined her. She was discharged improved after five days, with a diagnosis of *hysteria*.

GROUP 11.—*Psychopathoses*. Psychopathies, and the unclassified

It was Southard's intention that in this group there be included not only those cases of dubious mental pathology who are variously classified as "constitutionally inferior," "psychopathic personality," "defective delinquent," etc., but also those occasional instances of psychotic forms entirely aberrant from any recognized type or group. These cases in which there is no possibility of agreement on a comprehensive and justifiable diagnosis of any kind are relatively infrequent, but they are common enough to be well known in private practice as well as in institutional work.

The following instance is perhaps not the best example, but it serves in a measure because of the strong suggestion of psychopathy and because of the precipitation of symptoms by influenza, although the influence of the latter is equivocal. I am aware of the similarity in some respects of this case to those described by Grasset,⁴⁴ Krannhals, and in the German army reports, as mentioned by Leichtenstern¹⁶ and epitomized as "the so-called hystero-epileptic [sic] attacks, occasionally

44. Grasset: *Leçons sur la grippe*, Paris, Masson et Cie; Montpellier, Coulet, 1890.

associated with fits of crying." Nevertheless, I agree with the intimation therein put forth that the demonstration of the hysterical nature of those phenomena (even if this case be proved to be similar) is not wholly convincing.

CASE Q.—Psychopathic Basis (+?) + Influenza = Undiagnosed Psychosis.

Family History.—The patient was a married woman, aged 35, whose occupation was housework; she was born in Nova Scotia. Her grandparents' history was negative except that "patient states that she heard her father say that his mother lost her mind after her son had been killed." Both parents were living. Her father was 60, mother 66, father well, mother crippled by "rheumatism." Siblings, four living and well, three died in childhood (scarlatina, drowning, and "unknown"). No history of mental disease in family.

Past History.—She was born in Nova Scotia in 1884; full term, normal delivery. She passed a normal childhood, except for somewhat inferior physical status. "She was always the delicate one of the family." Had measles and scarlatina; later "chronic bronchitis." Was "always nervous."

Educational: There is some discrepancy in the accounts of her schooling, but at any rate she was a rather superior child and is said to have been in the eighth grade at the age of 13. She left high school at the end of the first year to get married. Her school work seems to have been very good, and she was not held back.

Economic: She worked for about a year just prior to marriage, at a Boston department store. Other than this she never did anything but housework.

Marital: Married at 16 to a private detective who never properly supported her and who eventually deserted her when her youngest child was 5 weeks old. She later obtained a divorce. He alleged immorality on her part (while married to him), which she denies. There were three children, all living and well, aged 16, 14 and 11. In addition there were several self-induced miscarriages.

Subsequent to her separation from her husband she was sexually intimate with two men, whose names she gives. These indiscretions had an important relation to her present illness.

Previous Nervous Trouble: Five years previously she was in the outpatient department, because of insomnia, worry over children who were in custody of divorced husband at that time, paresthesias of legs, tingling in hands, supposed loss of memory, inadequacy, etc. Given a few hydragric treatments; improved.

Character: Not seclusive. Not much adequate information secured because of prejudice of informants (including patient).

Present Illness.—Influenza began October 9. She was ill only two days. "Following this, however, she was unable to sleep and she could not think right. She has great difficulty in recalling what she did the next few days. Then she commenced worrying over her recent sex indiscretions. To add to her troubles a young niece died of influenza on the 12th."

She was brought to this hospital on the 18th by her father. The admission note reads as follows:

"In admission office the patient at first refused to answer questions. Then said there was no reason for her coming here. Is afraid that she is going to be killed here. Does not show any marked emotion over it, however." Later in the ward, "she is pleasant and accessible, and does not appear apprehensive

or depressed. No hallucinations or definite delusions. Says she had influenza following which she became depressed and retarded; now complains of feeling weak."

Mental Examination.—*Orientation* precise in all spheres. *Memory* intact except for events just preceding admission, which are apparently quite hazy. *Ideation* characterized by self-accusatory ideas; for example, she had disgraced her children by her sexual indiscretions; the nurses knew of her miscarriages from her chart and would look down on her; father and mother would find out about her, or be informed of what she had done. There was no *insight*, nor on the other hand any definite delusions. *Hallucinations* absent. Thought processes showed no irrelevancy, incoherency or retardation. *Emotional Tone*: Except for periods as described below, her emotional tone seemed to be quite normal; she was cheerful and pleasant and responded to interrogation freely and cooperatively. The attacks described were accompanied by wailing and followed by singing; it was impossible to determine what or how she felt emotionally.

Motor Status.—The characteristic feature in the case was the frequent outbursts of wailing, shrieking and weeping, accompanied by a clonic stiffening of the muscles of her entire body, and a flushing of the face. These attacks were of perhaps twenty minutes' duration; they were sometimes induced by interrogation on the matter of her indiscretions, but at other times she answered the same questions with no disturbance whatever. The manifestations differed slightly at different times, but the essential things were complete inaccessibility because of a lugubrious demonstration which was in some cases followed by equally inexplicable elation. At one time during an attack, when she was stiffened out tensely, a pinprick test was attempted and she was found to be anesthetic.

She herself explained these attacks on the basis that she "felt so degraded" to think she would be suspected of immorality. Yet this she freely admitted, and at the same time insisted that she was sexually frigid.

Still another type of conduct disorder appears in the following interrogation, which also gives a good idea of her type of response: (One night you did a great deal of screaming, and pounding on the door.) "No, it was morning."

(What time?) "It was after breakfast. I had spent an awfully restless night and I went in and asked Miss P. if I could lie down. She just took me and threw me into the cells there, and I got into a kind of frenzy."

(We have no cells here.) "I thought it was locked. She slammed the door so hard that I thought she locked it."

(Even if she had locked it?) "I got awfully frightened. I have a home and three children and I want to go home to them."

(Are you afraid to be left alone with yourself?) "No, I am perfectly safe alone."

(I don't see why you should be so frightened?) "Just a terror came over me."

Physical Examination.—General hyperalgesia. Positive Romberg. Reflexes normal, except knee jerks which, although equal, were diminished. Blood pressure, systolic 118, diastolic 62.

Laboratory Findings.—Urine, blood serum Wassermann test, spinal fluid (including Wassermann test), vaginal smear and complement test for tuberculosis all negative.

Psychometric Test.—The patient graded regularly at a mental age of 18. In the supplementary tests her performance in the construction puzzles was

good. The memory tests were also well done. She showed fairly good comprehension and judgment in the apperception picture puzzle. She *accepted* two out of ten *suggestions* [sic]. She cooperated well.

She showed some improvement, but was committed.

The diagnoses made here ran the gamut of psychiatric possibilities, including dementia praecox, psychopathic personality, and psychoneurosis. Against the first named is preeminently the absence of schizophrenia. Against psychoneurosis there is less definite proof, but it might be pointed out that her conduct was much disturbed, that insight was completely lacking, that it was not proved that she was hypersuggestible or bore stigmata of hysteria at all, and finally that she was committed. The possibility of cyclothymic psychosis may be suggested, but the absence of any constant depression or elation, and the absence of retardation at all times, makes this rather unlikely.

SUMMARY

The effect of influenza on the brain may now be considered analytically by summarizing the cases collected and detailed in the foregoing.

There are obviously two methods of approach: One may consider in summary merely the end results, which being the matter of primary pragmatic value is usually considered first. Complementary to it, however, is the study of the component essentials, the basis and contributing factors which together bring about the final product, a study of the material on which the influenza acted to produce specific results.

Considering the first method, one may tabulate the paradigms to show the end results thus (in Southard's order):

Apparent Normality + *Influenza* = *General Paresis (Neurosyphilis)*.

Morosis + *Influenza* = *Imbecility (Hypophrenia)*.

Epilepsy + *Influenza* = *Alteration in Frequency and Type of Epilepsy*.

Alcohol(ism) + *Influenza* = *Delirium Tremens (Etc.) (Alcoholic Psychoses)*.

Normality + *Influenza* = *Cerebral Hemorrhage (and Psychosis)*.

Normality + *Influenza* = *Delirium (simple, errant, schizophrenic, etc.)*.

Senescence + *Influenza* = *Senile Psychosis*.

Normality } + *Influenza* = *Dementia Praecox (Schizophrenia)*.
or
Predisposition }

Normality } + *Influenza* = *Manic Depressive (Cyclothymic) Psychosis*.
or
Predisposition }

Normality } + *Influenza* = *Psychoneurosis (Hysteria, Psychasthenia, etc.)*.
or
Predisposition }

Psychopathy + *Influenza* = *Psychosis (Atypical)*.

From this presentation one is justified in the conclusion that any form of mental disease may follow influenza. But this is to declare a chronologic relationship only, and to imply or assume an etiologic connection that only a study of the component elements of the equation can justify or elaborate.

In a sense, we should be justified in urging in place of the phrase "influenzal psychoses," which literally do not exist, the more cumbersome but more correct expression, "influenzal neurotoxic effect (or product)." Surely the most striking and noteworthy fact is the variety of psychic (and encephalopathic) lesions. This fact alone is particularly and specifically intolerant of a doctrine of specificity. This we may say without reference to the modifying or even determining factors contributive in each case or group of cases, simply because of the great inclusiveness of the list. A disease—an incident—which is capable of calling forth psychiatric pictures as widely different as general paresis and hysteria (I temporarily waive the point that in neither case cited as illustrating these phenomena was there any previous indication of mental trouble), and which does not fail to leave its conspicuous impression on every intervening group of mental disease in a representative list, can surely not longer be accused of possessing psychic specificity.

Reversing the point of emphasis, now, and considering minutely the left-hand side of the equation, the components of the sum, we may tabulate the cases in three groups. Primarily, there are those arising on a basis of undisputed psychic normality. In these the left-hand side of the equation reads throughout:

$$\text{Normality} + \text{Influenza} =$$

These are distinctly psychoses of *creation*, as opposed to psychoses of precipitation, revelation or alteration. I have suggested these terms as descriptive of the process involved merely as useful figures, and without presuming to indicate what psychopathologic process actually takes place. It is established that this is what *seems* to take place, and these then are the processes that seem to be represented. The first one, *creation*, is represented by the following paradigms:

$$\text{Normality} + \text{Influenza} = \text{Delirium.}$$

Cerebral Hemorrhage with Psychosis.

Senile Psychosis (?)

Schizophrenia (Dementia Praecox).

Cyclothymia (Manic-Depressive).

Hysteria (Psychoneurosis).

The next group of equations are those in which the influenza acts on an avowedly predisposed soil, resulting in the production of a

psychosis of a specific form, exactly analagous to those mentioned in which no evidence of predisposition was shown to exist. These cases are based on equations the left side of which is uniformly

$$\textit{Predisposition} + \textit{Influenza} =$$

They represent a process to which scores of verbs have been applied. The French and German terms are scarcely less numerous than the English, and "provoked," "incited," "instigated," "produced," "called forth," and "excited" are only a few of these. The inference is that the nature of the process is frankly not understood. If I prefer to use the term "precipitated," it is not with the idea that it expresses any better conception of the nature of what takes place. Elsewhere there will appear shortly a more presumptuous attempt to liken the process to that of catalysis in chemistry, but I do not urge this here. This group, representing the process, we will say, of *precipitation*, is represented by the following paradigms in the above cases:

$$\textit{Predisposition} + \textit{Influenza} = \textit{Delirium Tremens (etc.)}.$$

Schizophrenia.

Cyclothymia.

Psychoneurosis.

Finally, there is a smaller group representing the power of influenza to alter the nature or degree of a neuropathologic or psychopathologic lesion already extant. These cases may masquerade under guises suggestive of a process primarily of revelation, such as the revelation of a heart lesion by an attack of pneumonia. Thus the appearance of general paresis immediately after influenza might simulate this, but actually it probably amounts to but an augmentation or hastening of a process no doubt already established, and possibly capable of detection were all the refinements of diagnosis possible of application in an unsuspected subject. This group, representing the power of influenza in the *alteration* of pathologic brain processes, is represented here by

$$\textit{Morosis} + \textit{Influenza} = \textit{Imbecility}.$$

$$\textit{Epilepsy} + \textit{Influenza} = \textit{Alterations in frequency and type}.$$

$$\textit{Psychopathy} + \textit{Influenza} = \textit{Psychosis}.$$

$$\textit{Apparent Normality (Latent Neurosyphilis)} + \textit{Influenza} = \textit{General Paresis}.$$

$$(\textit{Mild Neurosyphilis}) + \textit{Influenza} = \textit{Advanced Neurosyphilis}.$$

Influenza thus acts apparently in a nonspecific manner to do specific things; to create psychoses, to precipitate them in predisposed persons, and to augment or alter them where already existent. So powerful and so versatile a neurotoxin is certainly not possessed by any other acute infection. It is conceivable, although scarcely so, that were there

some other potent infection as ubiquitous as influenza, its neuropsychiatric effects might be as numerous and as various, but certainly evidence does not favor this. It is not true of the sequelae of the great plagues of typhoid and bubonic plague that once raged; lobar pneumonia is certainly common enough and widespread enough to have resulted by this time in a similar host of nervous sequelae were it akin to influenza in neurotoxic effect. Quantitative specificity, in point of high potentiality in neurotoxic effect, is apparently fairly claimed by influenza.

Finally, the writer would add a word in defense of the equation paradigms as used in this article. I am well aware, of course, that it is impossible to accurately and wholly express any biologic process in chemical or mathematical formulas. They have been adopted here, not for expressing the whole truth, even if anyone grant that to be attainable, but the essential truth as concerned with influenza, the human basis on which it works and the psychosis which succeeds. There are doubtless many elements entering into the formulas expressing influenzal action on human brain which are not represented in the paradigms of this article, elements which while absolutely indispensable to the complete configuration of the process, are in no way essential to the basic principles represented by the simpler and wholly pragmatic cryptograms.

CONCLUSIONS

1. This paper aims to illustrate the forms of psychoses associated with influenza by the presentation of representative cases.

2. The history of the study of the association of mental disorder and influenza is traced roughly from its inception in 1385 to its frank recognition in 1790 and its elaboration since then.

3. Southard's Eleven-Group nosology is commended by its mechanical convenience and its neat inclusiveness, and serves admirably to present cases illustrative of findings as follows:

4. Active neurosyphilis (Southard's Group 1) may be precipitated by influenza (and cases of sluggish course accelerated). (Case A.)

5. Hypophrenia (Group 2) may be augmented in degree, and a case of the apparent process "Morosis + influenza = Imbecility" is cited, but no evidence was obtained for the production of total loss of intellect, the acute dementia of the idiocy type, or Kraepelin's misnamed "infectious idiocy." (Case B.)

6. Epilepsy (Group 3) may be altered quantitatively and qualitatively, that is, in the frequency and in the form of attacks, but there were no instances of its initiation by influenza in our series. (Cases C and D.)

7. Delirium tremens and other forms of alcoholic psychoses (Group 4) were quite frequently induced by the added toxemia of influenza, but probably in no greater frequency than would obtain in a similarly large number of any acute infectious disease. (Case E.)

8. Of the encephalopathic psychoses (Group 5) the occurrence of Leichtenstern's influenzal "hemorrhagic encephalitis" with a peculiar psychosis was demonstrated clinically and by necropsy. (Case F.)

9. Delirium (Group 6) remains the most polychromatic and versatile of mental disease pictures; its association with influenza is notoriously frequent, and its manifestations bewilderingly multiform. It stands as the type illustration of the paradigm, "*Unknown factor + influenza = psychosis.*" (Case G.)

10. Of psychoses associated with senility and the presenium (Group 7) one rather equivocal case is presented as having been initiated by influenza without previous indications. (Case H.)

11. Schizophrenia (Group 8), cyclothymic psychosis (Group 9) and psychoneurosis (Group 10) occur following influenza with and without predisposition or previous manifestations. Instances of all are given in detail (Cases I, J, K, L, M, N, O and P.)

12. Undiagnosed psychoses and psychopathias (Group 11) form rather too vague a group to be considered categorically, but a representative case is given in which influenza incited a psychotic episode in a (?) psychopath. (Case Q.)

13. The cases presented may be summarized by paradigms, exemplifying the psychiatric effects of influenza, viz.:

(a) In the process of *Creation*:

Normality + Influenza = Delirium (simple, errant, schizophrenic).

Apoplexy, Atypical Psychosis.

Senile Psychosis (?)

Schizophrenia.

Cyclothymia.

Hysteria.

(b) In the process of *Precipitation*:

Predisposition + Influenza = Delirium Tremens (etc.).

Schizophrenia.

Cyclothymia.

Psychoneurosis.

(c) In the process of *Alteration*:

Morosis + Influenza = Imbecility.

Epilepsy + Influenza = Alterations in frequency and type.

Psychopathy + Influenza = Psychosis.

Apparent Normality (latent neurosyphilis) + Influenza = General Paresis.

*(Mild Neurosyphilis + Influenza = Advanced Neurosyphilis).**

14. Influenza apparently acts on the brain in three ways: to create psychoses, to precipitate psychoses in predisposed subjects, and to augment or alter their form where already existent.

15. Thus we cannot from the present data regard influenza as capable of *qualitative* psychic specificity.

16. The *quantitative* specificity, however, of the influenza neurotoxin is confirmed by its remarkable potency and versatility; the large number and wide variety of psychic and encephalopathic lesions produced being one of the most striking neuropsychiatric features of the epidemic.

17. The question of predisposition is simultaneously answered, there being evidence to show that psychoses sometimes occur directly after influenza with no forerunning symptoms or signs, and sometimes occur then only after months or years of less pronounced manifestations.

* Mentioned but not detailed in this article.

SENSORY CHANGES IN PERIPHERAL NERVE INJURIES

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The present article is based on a study of peripheral nerve injuries for a period of one year, during a part of which time I was stationed in France, and the balance of the time in this country. A large number of the patients were examined soon after the injury; many of them a year or two after the operation and others at various periods after receipt of the wound.

1. THE SENSORY DISTURBANCES IN PERIPHERAL NERVE INJURIES

The sensory disturbances in peripheral nerve injuries do not always go hand in hand with severity of the injury. This probably depends on the anomalies often present in sensory distribution, anastomosis (especially between median and ulnar) and the location of nerves supplying adjacent areas. I frequently encountered complete sections—found so at operation—showing slight sensory disturbances; and often incomplete or partial injuries of a nerve with extensive sensory disturbances. The first applied especially to the musculospiral nerve, the second to the ulnar. The musculospiral nerve is preeminently motor. Occasionally only a small area of anesthesia will be found around or over the first dorsal interosseous space. At other times it extends to the forearm, encroaching on the musculocutaneous area. A slight injury of the ulnar nerve is liable to give extensive sensory disturbances, frequently passing beyond the typical ulnar area. Rarely, complete section causes anesthesia just over the dorsal surface of the fifth finger. A slight injury of the external popliteal nerve may give extensive areas of sensory disturbances, while complete section of the sciatic nerve may produce less extensive changes.

I have selected for the first part of this article ten cases which gave the syndrome of complete interruption, subsequently corroborated by operation. This syndrome was the result either of complete nerve section or interruption by scar tissue, fusiform growth, etc., so that nerve fibers, if present, were degenerated. Therefore, in this article "syndrome of interruption" means that no healthy and acting nerve continuity exists.

The following illustrations were used to represent the various sensory changes:

CASE 1.—L. P., received a gunshot wound in the outer side of the thigh; the upper third. Findings: The sciatic nerve was found with a large bulbous enlargement 5 cm. below the gluteal fold. Faradic stimulation below the bulb did not elicit response. Operation was performed five months after injury. Two months after operation there was no motor improvement. Sensory changes were as indicated in Figure 1.

CASE 2.—H. L. received a gunshot wound, antero-internal aspect; the lower third of the left thigh. Findings six months after injury: The sciatic nerve was bound down by dense scar tissue. The internal popliteal portion showed a bulb for a distance of about one-half inch. Sensory Figure 2 was obtained several days before operation. The existence of a small area of hyperalgesia made the diagnosis of a complete section doubtful.

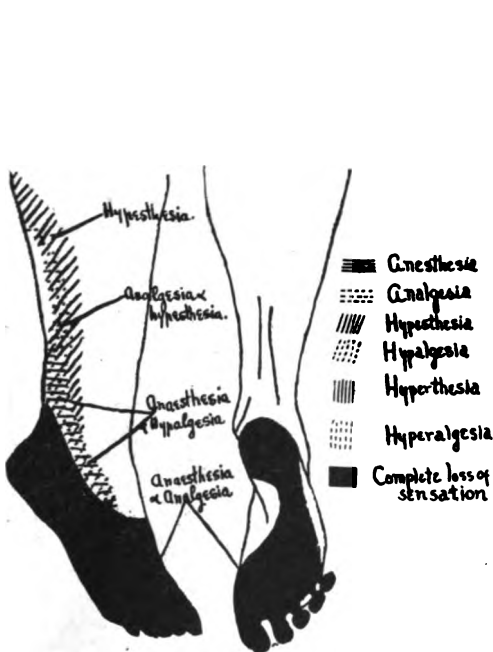


Figure 1

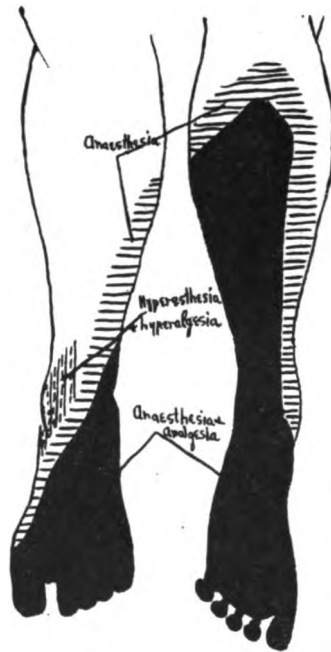


Figure 2

Fig. 1 (Case 1).—L. P., peripheral nerve injuries following gunshot wound of the thigh.

Fig. 2 (Case 2).—H. L., peripheral nerve injuries after gunshot wound of the thigh.

CASE 3.—F. J. received a gunshot wound, antero-external surface; the middle third of the left arm. Findings at operation five months later: The spiral nerve was separated for a distance of $1\frac{3}{4}$ inches. Sensory Figure 3 was made several days before operation.

CASE 4.—C. C. suffered a wound of the right axilla due to a hand-grenade explosion. Findings: The median nerve was separated for a distance of one-half inch. The ulnar nerve could not be freed from scar tissue. Examination was made ninety-six days after nerve suture. Regeneration of the median nerve had begun, but there was no improvement in the ulnar. Clinically we had the syndrome of complete interruption of the ulnar nerve (Fig. 4).

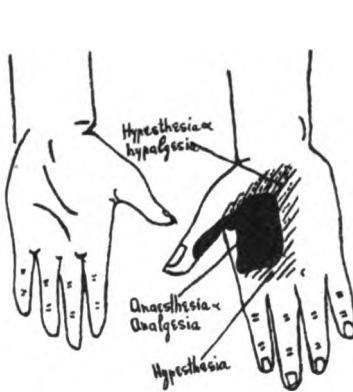


Figure 3

Fig. 3 (Case 3).—F. J., nerve injuries resulting from gunshot wound of the arm.

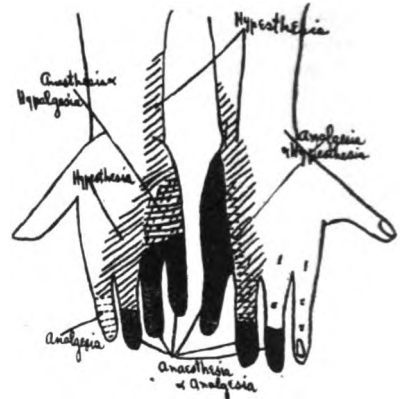


Figure 4

Fig. 4 (Case 4).—C. C., nerve injuries from a hand-grenade wound of the right axilla.

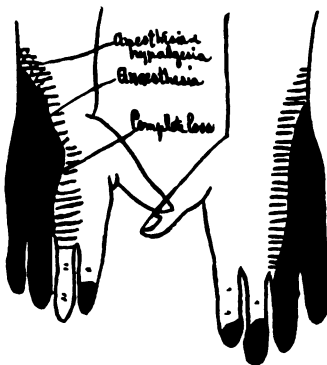


Figure 5

Fig. 5 (Case 5).—L. J., nerve injuries following a gunshot wound of the arm.

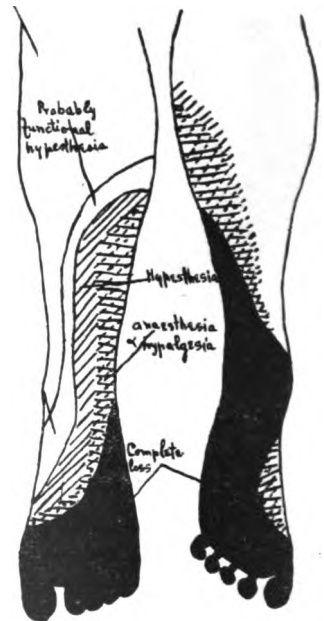


Figure 6

Fig. 6 (Case 6).—Z. A., nerve injuries after high-explosive wound of the thigh.

CASE 5.—L. J. suffered a gunshot wound, anterior aspect; lower part of the arm. Findings at operation ten months later: The ulnar and median nerves were divided. The ulnar was separated for a distance of three-quarters inch, and the median for $2\frac{1}{2}$ inches. Figure 5 was made one month before operation. The minor sensory changes in the median area, and extensive sensory disturbances in the ulnar region should be noted.

CASE 6.—Z. A. suffered a high-explosive wound of the lower third of the left thigh. Findings at operation six months after injury: The external and the internal popliteal nerves were divided, presented large bulbous ends and were firmly bound down in scar tissue. Figure 6 was made five months after injury. The extensive sensory changes may be noted. In this patient the Tinel sign was present and also formication in toes.

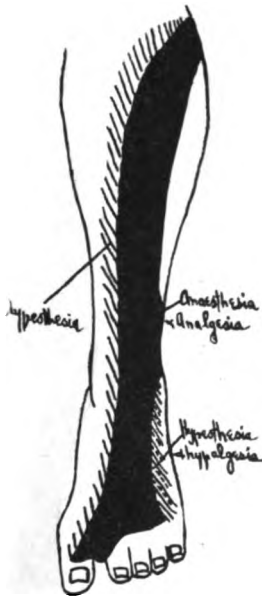


Figure 7

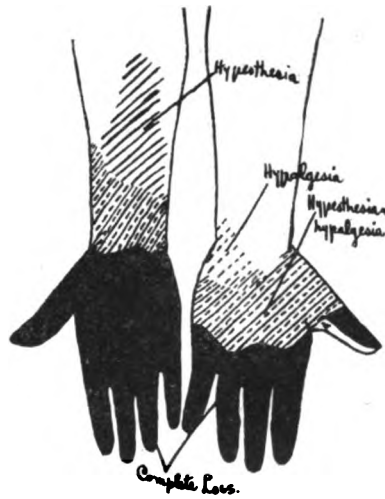


Figure 8

Fig. 7 (Case 7).—L. S., nerve injuries resulting from a gunshot wound across the popliteal space.

Fig. 8 (Case 8).—G. W., nerve injuries from high-explosive wound of the arm.

CASE 7.—L. S. received a gunshot wound across the popliteal space. Findings at operation nine months after injury: The external popliteal nerve was divided, with a large bulb at both ends.

CASE 8.—G. Wm. suffered a high-explosive wound, internal aspect of the right arm, the middle third. Findings at operation ten months after injury: The ulnar and median nerves were divided and separated for a distance of $1\frac{1}{2}$ inches. Figure 8 was made one month earlier. Extensive sensory changes in musculospiral area, no doubt due to functional disorder, should be noted.

CASE 9.—W. T. suffered a high-explosive wound, middle of the left forearm. Findings at operation nine months later: The ulnar nerve was divided

and separated for a distance of 3 inches. Figure 9 was made one month before operation. The slight sensory changes as compared with those in other ulnar nerve sections should be noted.

CASE 10.—V. J suffered a high-explosive wound of the right axilla. Findings at operation seven months after injury: There was a very large mass of glands enveloping the nerves just below the cords of the plexus. The median nerve was divided with a 5 cm. defect. The internal cutaneous nerve was divided with an 8 cm. defect. There was a small amount of scar tissue around the musculocutaneous and musculospiral nerves. The ulnar nerve was divided

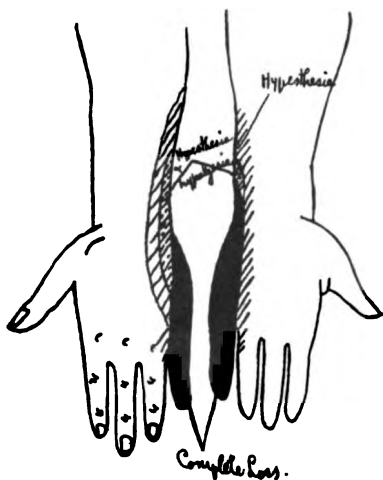


Figure 9

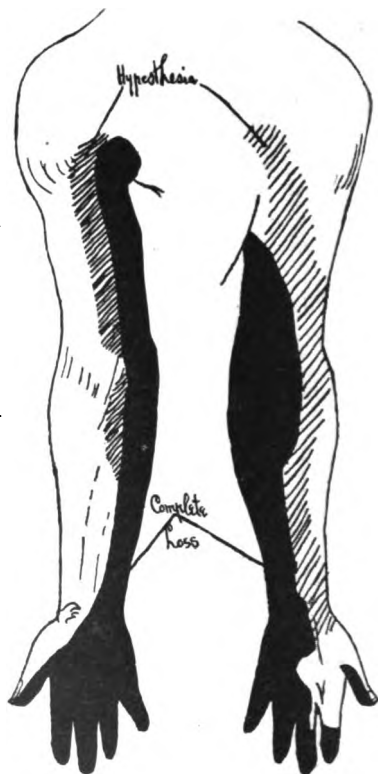


Figure 10

Fig. 9 (Case 9).—W. T., nerve injuries following a high-explosive wound of the forearm.

Fig. 10 (Case 10).—V. J., nerve injuries after a high-explosive wound of the right axilla.

with a 10 cm. defect. Figure 10 was made a month before operation. The patient had a positive Tinel in the median nerve and formication in the median area. The musculospiral nerve showed no changes.

All the foregoing operations were performed by Dr. Charles A. Elsberg at the U. S. Army General Hospital No. 1, Williamsbridge, N. Y.

COMMENT

In testing for loss of sensation of touch I used a brush with long, soft hair, and it was found necessary to have the parts shaved. In testing for analgesia, I used a pin, and it was necessary to distinguish between pressure and pain. Frequently the patient would say that he felt the pressure but not the prick of the pin. In the syndrome of complete interruption anesthesia and analgesia were always present, and as a rule I found the area of the former to be more extensive, although occasionally the reverse was true. In determining the threshold stimulus and the localizing power, I employed a pair of compasses with blunt points. The results obtained were not alike in all the nerves, but were about the same in injuries of the external popliteal, sciatic and ulnar nerves.

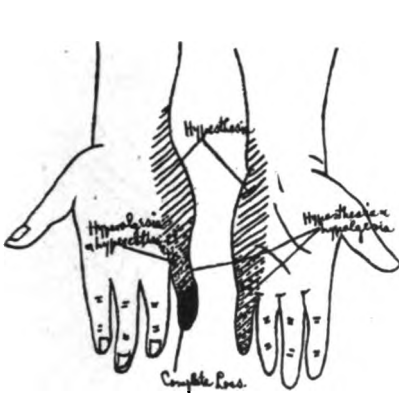


Figure 11

Fig. 11 (Case 11).—H. H. J., nerve injuries resulting from a high-explosive wound of the forearm.

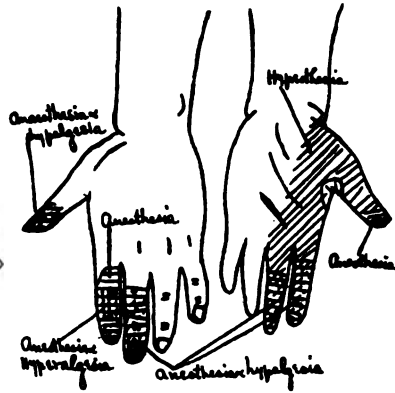


Figure 12

Fig. 12 (Case 12).—L. J., nerve injuries from a high-explosive wound of the forearm.

Hypesthesia and hypalgesia were always present in complete sections, comprising a zone about the area of complete sensory loss. On the other hand, a hypesthetic area was not always hypalgesic, and vice versa. Examination of the figures will show that there are areas where hypesthesia was present and the sensation of pain was normal.

The sensation of heat and cold was generally abolished, but more often that of heat. Warm felt as cold, or cold as warm was a rare finding.

In the syndrome of complete interruption, areas of hyperesthesia and hyperalgesia were never encountered. Whenever such areas were found the diagnosis of complete section was doubtful.

2. SENSORY SIGNS OF REGENERATION

After investigating a great many cases, I came to the conclusion that the Tinel sign of regeneration possesses a corroborative value only. It was present in cases of complete nerve section with no regeneration. In Case 10 the median nerve was found to be divided, yet the sign was present. The same is true of Cases 2 and 6. I know no reason for its presence in such cases. In ulnar and median lesions it is necessary to question the patient closely as to the exact location and extent of the sign. Occasionally in ulnar lesions it was present in the median area. This could be explained either by ulnar and median

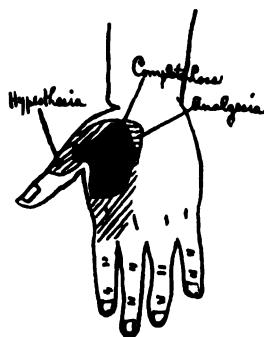


Figure 13

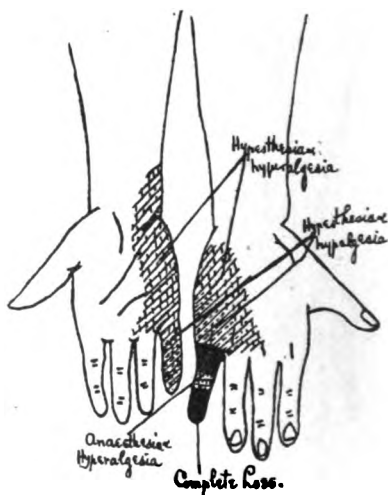


Figure 14

Fig. 13 (Case 13).—D. T., nerve injuries following a high-explosive wound of the elbow.

Fig. 14 (Case 14).—F. H., nerve injuries after a high-explosive wound of the forearm.

anastomosis, or by transmission of the pressure or tapping to the median, which had suffered a slight injury and was now regenerating.

In injuries of the sciatic nerves, when only one trunk, especially the internal popliteal, was injured, the formication was oftentimes present in the external popliteal distribution.

Examination of a number of postoperative cases and cases of spontaneous recovery revealed the fact that sensation returns before motion. A study of the figures 11-17 will show the nature of the changes in: (1) partial lesions with no operation; (2) postoperative cases; (3) cases of spontaneous recovery.

CASE 11.—H. H. J. suffered a high-explosive wound, middle third, antero-internal aspect of the left forearm. Examination eight months after injury: Syndrome of partial interruption of ulnar nerve, with signs of regeneration (Fig. 11).

CASE 12.—L. J. suffered a high-explosive wound of the postero-external surface of the left forearm, middle third. Examination 203 days after injury: Syndrome of partial interruption of the left median. The Tinel sign was present. The extent of hypesthesia over palm of hand should be noted (Fig. 12).

CASE 13.—D. T. received a high-explosive wound, internal surface of the left elbow. Examination 121 days after injury: Syndrome of partial interruption of musculospiral nerve. The Tinel sign was present. The absence of sensory changes on the palmar surface of the hand should be noted (Fig. 13).

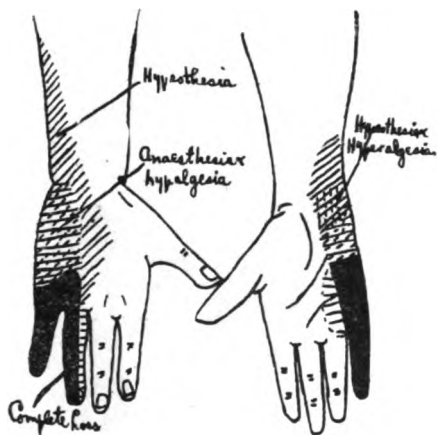


Figure 15

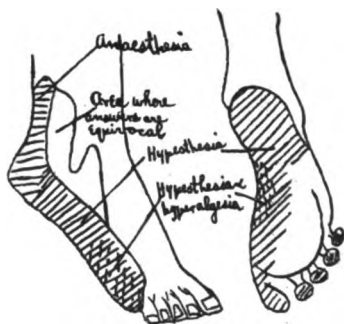


Figure 16

Fig. 15 (Case 15).—A. A., nerve injuries resulting from a high-explosive wound of the forearm.

Fig. 16 (Case 16).—C. F. W., nerve injuries from a machine-gun bullet wound of the thigh.

CASE 14.—F. H. suffered a high-explosive wound, anterior aspect, middle third of the right forearm. There was a syndrome of partial interruption of the ulnar nerve. The sensory figure was made 179 days after the injury. His history indicated that he had had also an injury to the median nerve from which he had recovered. At the last examination he had a very pronounced Tinel sign in the ulnar and median nerves. The only sensory disturbance in the median area was hyperalgesia on the upper palmar surface of the index finger (Fig. 14).

CASE 15.—A. A. had a high-explosive wound of the middle surface, internal aspect of the right forearm. The sensory figure was obtained 175 days after injury. The Tinel sign was present. There was a syndrome of partial interruption of the ulnar nerve. The area of anesthesia extended into the region supplied by the internal cutaneous nerve (Fig. 15).

CASE 16.—C. F. W. received a machine-gun bullet wound, anterior aspect of the right thigh. Examination 150 days after the injury detected a slight weakness in the distribution of the internal popliteal nerve. The Tinel sign was positive (Fig. 16).

CASE 17.—W. D. suffered a high-explosive wound of the internal surface of the upper third of the right arm. Examination 165 days after the injury disclosed in addition to a lesion of the musculospiral nerve, also a partial injury (slowly regenerating) and a musculocutaneous lesion. He had a strongly positive Tinel sign for the ulnar nerve. The musculocutaneous nerve in spite of the injury, did not show any motor loss, the only evidence of its injury having been the change in sensation.

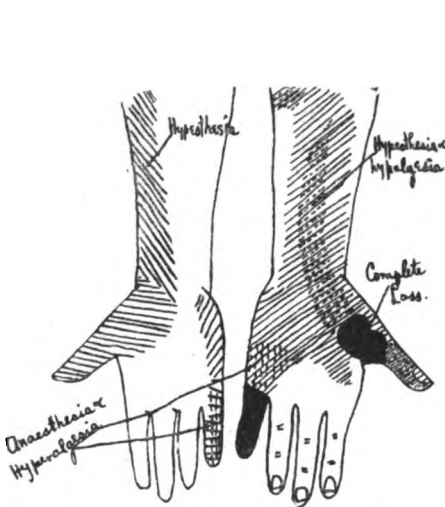


Figure 17

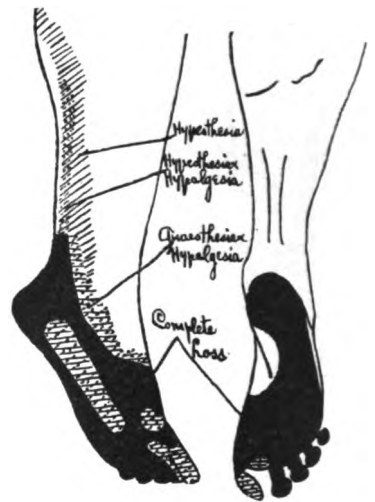


Figure 18

Fig. 17 (Case 17).—W. D., nerve injuries following a high-explosive wound of the arm.

Fig. 18 (Case 18).—L. P., nerve injuries after gunshot wound of the thigh.

The following patients are those who were operated on either on account of complete interruption or because of imperfect regeneration:

CASE 18.—L. P. This patient has been discussed under Figure 1. The present figure was obtained three months after nerve suture. It shows beginning regeneration. In the middle of the anesthetic and analgesic area, on the external surface of the foot, a small area of hypalgesia appeared, which in course of time would become hyperalgesic. The anesthetic area also became smaller. The figure also shows the independence of the fibers for superficial sensation of touch and deep sensation of pain. There were areas in which anesthesia was present with hypalgesia, and an area with analgesia and hypesthesia. The latter area was previously anesthetic and analgesic. In spite of the improvement in sensory symptoms, there was no motor improvement (Fig. 18).

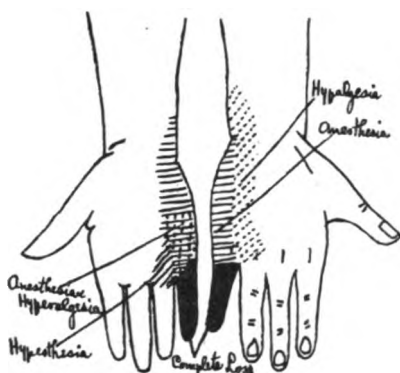


Figure 19

Fig. 19 (Case 19).—O. W., nerve injuries resulting from a high-explosive wound of the forearm.

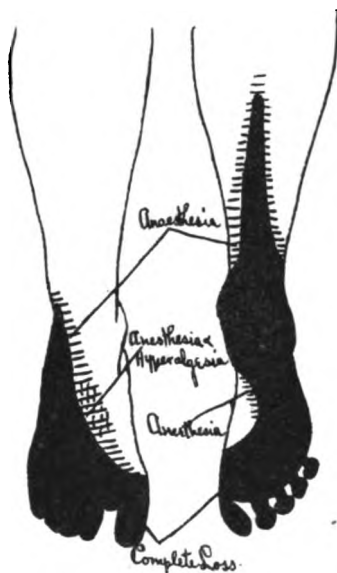


Figure 20

Fig. 20 (Case 20).—S. B., nerve injuries from a high-explosive wound of the thigh.

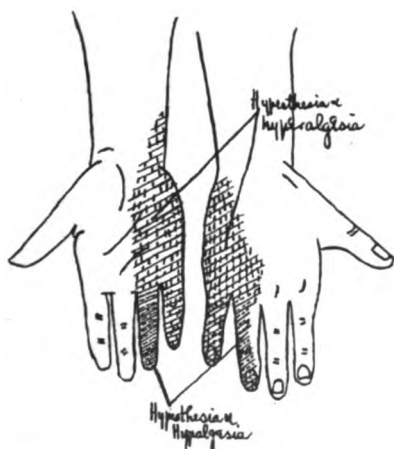


Figure 21

Fig. 21 (Case 21).—S. M., nerve injuries following a high-explosive wound of the forearm.

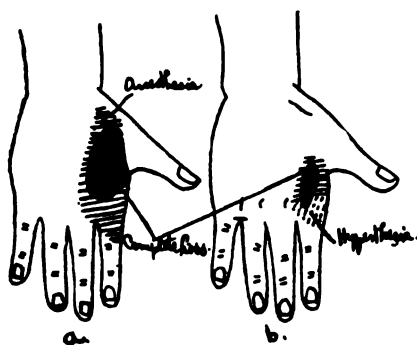


Figure 22

Fig. 22 (Case 22).—B. M., nerve injuries after a high-explosive wound of the arm. Motor improvement was more rapid than sensory.

CASE 19.—O. Wm. suffered a high-explosive wound of the internal surface of the right forearm, lower third. Examination was made ninety-three days after nerve suture for a complete ulnar. The Tinel sign was present. The improvement in motion was very slight, but sensory improvement was very marked. Muscular atrophy was very pronounced (Fig. 19).

CASE 20.—S. B. received a high-explosive wound of the posterior surface of the right thigh, middle third. The figure was obtained two months after nerve suture for a complete sciatic. There was no motor improvement but hyperalgesia and hyperesthesia had appeared. In this patient the Tinel sign was absent, and there were no signs of regeneration except the slight sensory change. He also showed very little muscular atrophy, much less than would be expected after an injury of about six months' duration (Fig. 20).



Fig. 23 (Case 23).—McT., nerve injuries resulting from a high-explosive wound in the popliteal space.

CASE 21.—S. M. had a high-explosive wound, internal aspect of the right forearm, lower third. He had a partial ulnar, regeneration not having taken place on account of mechanical obstruction. Small fragments of bone were removed. Figure 21 was obtained 173 days after operation. There was no motor improvement.

CASE 22.—B. M. This patient was the first one seen in whom motor improvement was more rapid than sensory. Figure 22 (*a* and *b*) shows the sensory disturbances before and after operation, only six weeks apart. The patient had a high-explosive wound, anterior surface of left arm, just above the elbow. A small piece of shrapnel was still present in the wound and probably interfered with regeneration of the musculospiral nerve, which showed partial injury. Figure 22*a* shows the sensory disturbances taken four months after injury. He was operated on one week after the examination, or five months after the injury, and the fragment was extracted. Thirty-nine days after operation Figure 22*b* was obtained. As can be seen, there was slight improvement in sensation; on the other hand, the improvement in motion was remarkable. All the movements of extension were possible, without limitation in the slightest degree.

COMPLETE SPONTANEOUS REGENERATION OF THE EXTERNAL
POPLITEAL NERVE

CASE 23.—McT. had a high-explosive wound in the right popliteal space. Examination seven months after injury disclosed a slight limitation in the dorso-flexion of the right foot and a suggestion of right toe drop. Examination of flexors and extensors of the foot showed no abnormality. Figure shows sensory disturbances (Fig. 23).

COMMENT

The first sensory changes appeared in my cases as early as thirty days after operation or after injury. In cases of spontaneous recovery it is impossible to say when the first changes appear. The musculospiral and the ulnar probably regenerate earlier than any other nerve. The first sign of regeneration of the sciatic nerve appeared as early as two months after operation.

The second phase was the reduction of the anesthetic and hypesthetic areas. Especially the anesthetic area became smaller and spots of hypesthesia appeared in the middle of the anesthetic zone. With the reduction of the analgesia there appeared patches of hyperalgesia. These become more marked in the course of time, until anesthesia and analgesia entirely disappeared.

It is important to test for vibration perception in such cases with the tuning fork. Hyperesthesia was not so frequently found as hyperalgesia. During the third phase of regeneration, analgesia and anesthesia disappeared leaving a small area of hypesthesia or hyperalgesia. Hyperesthesia and hyperalgesia are never present in complete interruption.

I am especially indebted to Major G. E. Price, M. C.; Major H. O. Feiss, M. C.; Lieut.-Col. E. G. Zabriskie, M. C., and to Dr. (formerly Major) T. H. Weisenburg for opportunities, stimulus and advice.

Abstracts from Current Literature

PATHOGENESIS OF DIPHTHERITIC PARALYSIS. F. M. R. WALSHE,
Quart. J. Med. **11**:191 (April) 1918; **12**:14 (Oct.) 1918; **12**:32 (Jan.) 1919.

This interesting paper, in three parts, first reviews the rather scant literature on the subject, then compares the mode of invasion of the nervous system in diphtheria to that in rabies, details the author's unusual experience in thirty cases of paralysis following extrafaucial diphtheria, and finally draws certain conclusions.

The author's cases were those of diphtheritic invasion of wounds and cases of so-called "barcoo rot" or "septic sores" which he shows may fairly be considered diphtheritic, although until recently their nature was thought to be obscure. His conclusions are;

1. Palatal paralysis does not occur except after faucial diphtheria.
2. The musculature of the palate and the region of the infective focus derive their innervation from the same source, the glosso-pharyngeal-vagus-accessorius nuclear system and its peripheral fibers, and are thus closely related anatomically.
3. Similarly, in extra-faucial diphtheria the paralysis often shows (27 per cent.) an onset anatomically related to the infective focus.
4. Polyneuritis follows both faucial and extra-faucial diphtheria equally, and irrespective of the site of the infective focus presents a remarkably constant symptom-complex.
5. Paralysis of accommodation also follows both forms of infection, though the faucial more frequently (86 per cent.) than the extra-faucial (33 per cent.). It is, therefore, not a "local" paralysis, and its more constant association with faucial infections may be explained by the close proximity of the local central nervous lesion in this case to the oculomotor nuclei. To this extent a "local" factor may be admitted.
6. The grouping of the symptoms of tetanus, adopted by Meyer and Ransom, into local, specific, and generalized, may be applied equally well to the nervous phenomena of diphtheria. The palatal paralysis constitutes the "local," the ocular affection the "specific," and the polyneuritis the "generalized" forms of diphtheritic paralysis. In extra-faucial diphtheria, as in tetanus, the "local" paralysis varies with the site of the infective focus.
7. As for the pathologic processes underlying this syndrome, it seems highly probable that as regards the local paralysis we are dealing with an "ascending lymphogenous toxi-infection" (by the perineural lymphatics) of the central nervous system from the infective focus. The essential pathologic lesion here is central and not peripheral, and is situated in the nuclear complex already described.
8. The ocular and generalized symptoms are probably the result of the circulation of the toxin in the blood stream, whence it gains access to the whole nervous system, central and peripheral. The essential nervous lesion here is probably both central and peripheral.

Dr. Walshe states that these conclusions require confirmation by experimental and pathologic investigations. In part three, however, he details a case of diphtheritic ulcer of the perineum which strongly supports his conclusions. This sore was followed first by anesthesia of perineum, anus and adjacent parts of buttocks and thighs, then by involvement of bladder and later by multiple neuritis. The anesthesia of perineum and thighs showed the ordinary saddle shape of sacral cord disease and the author thinks it was due to involvement of the second, third, fourth and fifth sacral segments.

PATRICK, Chicago.

THE OPERATIVE TREATMENT OF SPASTIC PARALYSIS (HEMIPLEGIA, MONOPLEGIA, PARAPLEGIA) IN GUNSHOT WOUNDS OF HEAD AND SPINAL CORD. PROF. FOERSTER, Breslau, Deutsch. Ztschr. f. Nervenhe. May 7, 1918.

The author describes the procedures followed in sixteen cases of spastic paralysis of various types following gunshot wounds of brain or cord.

The operation of choice in severe spastic paraplegia is resection of the posterior roots. This was done in two cases of this type according to the following technic: The dura was exposed from the eleventh dorsal spine to the second lumbar vertebra, with resection of two thirds of the posterior roots in the region of the lumbo-sacral enlargement. In the second case, the dura was exposed at the first operation and opened eleven days later with resection of two thirds of the posterior roots as in the first case.

In this exposure the roots are found lying closely together allowing approach through a comparatively small opening in the bony covering.

Not only was the spasticity completely and permanently removed, but the voluntary movement to a large extent returned.

In one case of severe paraplegia with spastic paralysis of both arms due to a wound of the head, resection of the posterior lumbar roots and the second sacral roots was followed by relief of spasticity, but voluntary movement was little improved owing, Foerster states, to the fact that the motor centers were destroyed at the time of the accident.

Not all paraplegias following injury to the uppermost part of both central convolutions are as severe as the cases noted above. The paralysis and spasticity may involve only both feet, while knee and hip joints are either entirely free or are only slightly spastic-paretic. The lesion is, in these cases, in the inner side of the right and left paracentral lobule. Here lies the center for the dorsal and plantar flexion of the foot while the center for the muscles moving the knee and hip joints is to be sought on the convexity of the same convolution. Several times spastic paralysis of both feet was associated with ataxia of both legs. In these cases the position sense in both legs was completely lost, the central lesion extending backward on both sides to the region of the parietal convolutions.

For spastic contracture affecting single muscle groups plastic operations on the tendons is to be recommended, care being taken not to injure the tendon sheath and synovial channels. Beside the tendo-achillis, lengthening of tendons may be done successfully on the flexor carpi radialis, palmaris longus, flexor carpi ulnaris, on the biceps flexor cruris, semi-membranosus and semi-tendinosus.

In muscles where a tendinous attachment is wanting, Stoffel's operation on the peripheral nerves may be used. The latter method is especially useful in spasticity affecting the quadriceps extensor and adductors in the lower extrem-

ity and the pronators, biceps, coraco-brachialis, brachialis anticus, etc., in the upper extremity.

In the long flexors of the fingers the Stoffel operation is to be recommended as simpler than the attack on the numerous tendons. The same holds true for the flexors of the toes. With the relief of the spasticity in the operated muscles voluntary movement returns in the antagonists.

Unfortunately, by this method the source of the spasticity is not removed; the centripetal impulses continue so that in severe spastic conditions there is a return of the condition. For a permanent result so many motor fibers must be resected that paresis results.

The following commonly occurring types of contracture deserve special consideration:

1. Contracture of the plantar flexors of the foot and the associated paralysis of the dorsal flexors.

2. The inclination of the foot to assume a position of supination and thereby to combine, under certain circumstances, with a marked inversion of the outer border during the supporting phase.

3. The "clawing in" of the toes in raising the foot and during the supporting stage.

4. Spastic contracture of the quadriceps and the associated paralysis of the flexors of the knee.

1. For the relief of spastic contraction of the plantar flexors of the foot lengthening of the tendo-achillis is given the first place. The entire sole of the foot rests on the ground, the gait improves and the power of voluntary movement returns in the dorsal flexors.

2. For the relief of the inclination of the foot to supinate during the attempt at dorsal flexion one of two methods may be employed. (a) The tibialis posticus may be partly paralyzed by resecting the nerve bundle to this muscle in the popliteal space. One quarter of the fibers are allowed to remain. (b) The tendon of the tibialis anticus may be split in its entire length, the separated portion being transplanted into the outer border of the foot.

3. To combat the tendency of the toes to become forcibly flexed when the foot is raised or during the supporting phase, the nerve bundle to the flexor longus digitorum is resected in the neighborhood of the posterior tibial nerve. In many cases with secondary retraction of the toe flexors tenotomy of the flexor tendons may be resorted to.

4. For the relief of spastic contracture of the quadriceps extensor femoris exposure of the femoral nerve and resection of a part of the bundle supplying each head of the muscle is recommended.

In general, there is found in the hemiplegic arm spastic contracture of the flexors of the fingers and a corresponding paralysis of voluntary movement in the extensors. In the thumb there exists contracture of the long flexors and the adductors with a corresponding paralysis of the extensors, abductor and opponens. In the wrist, in the majority of cases, there is a spastic contracture of the flexors and paralysis of the extensors. At the elbow, spastic contraction of the flexors and paralysis of the extensors is the rule. At the shoulder there is found spastic contracture of the latissimus dorsi and pectoralis major and paralysis of the elevators of the arm. The inward rotators are spastic and the outward rotators paretic.

The operative measures for the relief of these disturbances are as follows: The spastic contracture of the flexors of the fingers is best relieved by partial

resection of the fasciculi to the flexor profundus digitorum and flexor sublimis digitorum in the neighborhood of the median and ulnar nerves. At most only one third of the fibers are incised and then only after the fasciculi have been extensively split up so that the resection may not involve too many fibers going to any one tendon. The flexors of the thumb are dealt with in the same manner. Sometimes, to improve abduction the tendon of the flexor carpi radialis is transplanted into the tendon of the extensor brevis pollicis.

Adduction contracture of the thumb may be treated either by resection, in the palm of the hand, of the nerve to the adductor pollicis or by tenotomy of the insertion fibers of the adductor.

The spastic contracture of the flexors of the hand is best relieved by plastic lengthening of the palmaris longus and flexor carpi radialis. Care must be taken not to make the operation too extensive lest the contractile power be weakened. Their action is necessary for fixation of the wrist during the act of extending the fingers.

The nerve to the pronator radii teres is resected as it leaves the median nerve for relief of pronatory spasm. Resection of one third of the musculocutaneous nerve is done for the relief of contracture of the flexors of the forearm.

For contracture of the latissimus dorsi and pectoralis major muscles, Foerster formerly resorted to plastic lengthening of the respective tendons, but in order that the operation should be extensive enough it was necessary to invade the muscle tissue thereby leading, in most cases, to weakening of the muscles. He now exposes the brachial plexus and resects the nerves to these muscles.

Tenotomy of the subscapularis tendon for spasm of this muscle has also given way to partial resection of the subscapular nerves as they leave the brachial plexus.

The article is illustrated by photographs of cases operated on and by descriptive case histories.

INMAN, San Francisco.

DISMORFIE ENDOCRINE. F. GIANNULI, Riv. di antropol. 21:215.

The morphologic type of the individual and his deviation from the normal forms the basis on which the Italian school has oriented the study of endocrinology. From the clinical morphology started by De Giovanni a few decades ago, the author says, we have passed to the endocrine morphology which opens the horizon toward the origin of diseases and which should constitute an essential preparation to the modern clinic.

The author classifies the endocrine deformities into three great categories, namely, deformities of monoglandular type, of pluriglandular type and of dysglandular type. He observes that pure monoglandular types are not the cases encountered in practice. Two cases illustrating the first category and one case illustrating the third type are reported. The first two correspond to the fundamental types of eunuchs differentiated by Tandler and Gross, namely, the eunuchus gigas and the eunuchus lardaceus. The third is a eunuchoid in which the masculine sex is prevailing. The author points out that the same two types of eunuchs consecutive to extirpation of the sex glands may be found in primary diseases of the hypophysis. This fact indicates and proves the existence of intimate correlations between these two endocrine systems, correlations which have a great value in the genesic functions and which give to the hypophysis the same biologic importance ascribed to the sex glands.

CASE 1.—The characteristics of the first specimen, a man, 36 years of age were:

The height was 1.98 meters (6 feet 6 inches). The length of the lower extremities was 1.3 meters (4 feet 3 inches). There were thoracic deformities, the pelvis being large, almost feminine. There was complete absence of hair from the face, from the mons veneris and from the entire body. The penis, scrotum and testes were rudimentary and presented a rudimentary vulva opening. The voice was of the feminine type. Mentally the patient presents the hebephrenic type of dementia praecox and shows a tendency to passive pederasty.

CASE 2.—The second subject, a woman 19 years old, suffering from a chronic hydrocephalus, combined in herself three syndromes: one neurologic, one psychic and one endocrine—a clinical picture extremely rare. She menstruated at the age of 12, then the symptoms grew gradually worse. At the age of 14 a tetraparesis appeared together with a menopause praecocissima (as the author calls the early disappearance of the menstruation in this case). Since then the growth of the limbs stopped, which fact was accompanied by a rapid and abundant increase of fat. The physical impotence, which reduced the patient's life to a purely vegetative one, was associated with a marked mental deterioration which culminated in apathetic dementia. The main morphologic stigmata are: somatic infantilism, small hands and feet not proportional to the length of the limbs, fingers hammer shaped bilaterally; absence of hair from the body and from the pudenda which shows a very elementary constitution characterized by the absence of the labia minora and of the clitoris; vagina infantile.

The author, after illustrating these two fundamental types, makes a rapid review of the experimental and clinical work carried out on the sex glands and hypophysis by Pellican, Tandler and Gross, Erdheim and Stumme, Loris, Pende, Goldstein, Peritz, and others, and discusses the different theories on the correlations existing between these two endocrine systems. He admits that although the general correlations seem to be sufficiently proved, yet the specific ones are not proved, so we are unable to explain the physiologic and the physiopathologic mechanism operating in the interdependence existing between the hypophysis, its lobes, and the sex glands. Except in cases of cerebral diseases it is very difficult to determine which is the gland primarily involved in the case of a eunuch. Of the two cases reported by the author, the primary involvement of the pituitary may be clearly seen in the second case, while the pathogenic origin cannot be stated in the first case.

The author thinks that the first case was due to the primary lesion of the sex glands and expresses the opinion that the giantism is secondary to morbid processes of the testes while the primary lesion of the hypophysis leads to acromegaly.

After emphasizing the importance of the morphologic study of the subject as a necessary preparation to the modern clinic, the author advances the following conclusions:

1. The endocrine deformities best defined by experiment and by clinical experience are those which produce the type of the *eunuchus gigas* and that of the *eunuchus lardaceus* of Tandler and Gross.
2. There are eunuchs and eunuchoids originating from pituitary disturbances just as there are from experimental or pathologic castration in both sexes.

3. The pictures of the pituitary and gonadic eunuchoidism often are alike in the semeiologic effects. This fact proves that between the hypophysis and the sex glands exists a system of intimate endocrine relations which govern the sexual life and the skeletal development.

4. This important endocrine system governs the genetic function and the physiologic evolutionary and involutinal phases of the sex glands and also the diseases of the sexual apparatus have a histologic repercussion over it.

5. As several endocrine glands comprise the genesic endocrine system the eunuchoid deformities may be either chiefly monoglandular or pluriglandular in type.

6. As the genesic endocrine system may be subject to pathologic changes which manifest themselves somatically not with functional quantitative disorders but with qualitative disorders, there may be some aberrant eunuchoid deformities which present only vague analogies in monoglandular eunuchs and which belong to a third group of eunuchs, the dysglandular eunuchs.

SANTE NACCARATI, New York.

THE PROBLEM OF FATIGUE. R. A. SPAETHE, PH.D., J. Indus. Hygiene 1:22 (May) 1919.

This article is a comprehensive review of the literature on fatigue, the various phases of the subject being taken up in turn. Part 1 deals with the theories of fatigue, physiologic and psychologic. The author first summarizes concisely the chemical point of view, that the problem is one of energy transformation with a resulting "dynamic equilibrium;" that is to say, work that reduces the available energy to low potential, is equalized by rest, which restores it to a high potential. The results of experimentation on the isolated muscle are also discussed and the theoretical "dynamic equilibrium" is correlated with the experimental finding of a "fatigue level." This "fatigue level" is attained when muscular work is carried on at such a rate that the metabolic products, such as lactic acid, are removed from the muscle at the same rate as energy is brought to the muscle in the form of nourishment. These points are well illustrated by Figures 2, 3 and 4 (but Figures 3 and 4 should be more thoroughly explained, and Figure 3 should have an arrow showing the direction of the kymographic tracing). Sherrington's theory that fatigue is localized in the synapse is clearly put forward by a quotation from Ash, and the fact that "nervous activity appears to precede in an astonishingly economical fashion" is discussed.

The most interesting part of the paper to the neuropsychiatrist is the exposition of the theory of the "varying energy level." This is a conception long needed in physiology and medicine, and though promulgated in terms of introspective psychology by James and McDougall, it now seems at last to be brought into a working relationship with physiology and pathology. Cannon's work is cited to show that the adrenals may be looked on as the physiologic basis for such psychologic concessions as James' "reservoirs of power." The importance of the emotions is discussed at length, and it is shown that emotive stimuli by releasing epinephrin may "carry the human organism through critical periods," but that "if we draw too deeply and continuously on our reservoirs of energy we may develop a condition of hyperesthesia and ultimately suffer a complete nervous breakdown."

It is most gratifying to see the emotions given their full responsibility as a cause of nervous breakdown, and by such straightforward mechanistic theories only can we lay a basis for a dynamic psychology; but when the author

states that—"from this point of view, nervous breakdown would really be a condition of adrenin (epinephrin) poisoning"—we must take issue with him. It is probable that certain kinds of breakdowns do occur commonly in this way, especially the conditions of hyperesthesia and the anxiety states arising from environmental conditions (such as war), but "nervous breakdown" is a broad term and it must not be forgotten that many breakdowns have as their outstanding features lack of emotion, energy and satisfaction. (And just such cases as these have gone to war and thrived on the abundant stimuli of the new environment.)

In the second part of the paper the tests for fatigue are discussed, and it is shown that no one test can be satisfactory because of the complex nature of fatigue. The relation of fatigue to efficiency is shown to be less simple than most statisticians believe. Here again the "emotive stimulus" is given its proper value. The war experiences in speeding up industrial output are cited as cases in point. Here strong emotional stimuli were used over long periods and resulted in some breakdowns and in a widespread industrial unrest. In the author's words: "The problem of industrial fatigue boils down then, to the practical question of detecting the signs of incipient psychoneuroses." It is, however, important to understand that industrial fatigue does not arise from simple overwork but from returning to the job day after day, having *rested insufficiently* between the hours of work. Thus industrial psychology comes back to the problem of the individual. What a pity that Carleton Parker could not have lived to see his theories taken up by the biologists.

Examples of increased output following the shortening of working hours are given, and "scientific management" is justly criticized. The paper ends with a bibliography of eleven pages, showing with what painstaking care the author compiled his facts.

COBB, Boston.

PITUITARY DISTURBANCE FROM ADENOIDS. P. CALICETI, *Pediatrics* 27:161 (March) 1919.

Caliceti recalls the relationship of the pituitary to the adenoids and the damage that may be done to it by the latter. He reports two cases that bring out these facts and that show quite forcibly the great importance of early removal of the adenoids before damage has been done to the pituitary, followed by the well-known changes in body and mind. Beside the operative procedure, he finds pituitary feeding beneficial.

FRANTZ, New York.

SUGAR TESTS IN HYPERTHYROIDISM AND OTHER ENDOCRINAL DISORDERS. JOHN R. WILLIAMS and ELEANOR M. HUMPHREYS, *Arch. Int. Med.* 23:537 (May) 1919.

These authors state that the glucose tolerance and utilization test proposed by Hamman and Hirschman (*Arch. Int. Med.* 20:761 [Sept.] 1917) and as later modified by Janney (*J. A. M. A.* 70:1131 [April 20] 1918), is a useful procedure to differentiate those metabolic disorders in which traces of a reducing substance are excreted in the urine. This test is superior to others that depend solely on the determination of urine sugar as a means to measure the degree of disturbance in carbohydrate metabolism in hyperthyroidism and other endocrinal disorders.

ARMSTRONG, Katonah, N. Y.

CEREBRAL HEMORRHAGE OF THE NEW-BORN. MARGARET WARWICK,
Am. J. M. Sc. **158**:95 (July) 1919.

This paper is based on a study of necropsy findings. Little has been written on this subject since 1885, at which time Sarah McNutt established the relationship between cerebral hemorrhage at birth and Little's disease.

The study of necropsies reveals that intracranial hemorrhage is found in from 12 to 32 per cent. of cases. The location varies, the hemorrhage being subdural, subarachnoidal, intracerebral, intraventricular, etc. The most frequent location is over the cerebrum because the veins in this area are afforded little protection, and in rapid labor they have little chance to mold themselves and are ruptured by the over-riding of the parietal bones.

The author states that punctate hemorrhages of the epicardium and parietal pleura are always found associated with intracranial hemorrhage, and in many instances there is a general hemorrhagic diathesis. Partial atelectasis of the lung is almost always associated with cerebral hemorrhage which might be a contributory and not a primary condition. There is usually hemorrhage without softening of the brain substance, death usually occurring before these changes take place.

The causes of cerebral hemorrhage of the new-born, as given by many authors, are:

(a) Long interval of time between birth of body and birth of head. (b) Hemorrhage following asphyxia caused by twisting of the cord about the neck. (c) Too great pressure of perineum. (d) Severe labor pains causing pressure on the sides of head, which increases long axis and presses brain forcibly against the tentorium tearing its radiating fibers. (e) Incomplete or too quick molding resulting in rupture of veins.

The author after discussing these theories and study of individual cases concludes that there are three primary causes: (1) Traumatism of all kinds at birth; (2) congestion or stasis with rupture of vessels, that is, due to malpresentation, overgrowth of child, twins, etc.; (3) diseased condition of child in intra-uterine life with no relationship to labor, that is, hemorrhagic diseases of the new-born, prematurity, syphilis, congenital heart diseases and other toxemias.

Cerebral hemorrhage of the new-born is not brought about by one single cause but by an interrelation and interaction of a varying number of causes found in the complex circumstances governing labor.

DELONG, Philadelphia.

MORRHAGE OF THE NEW-BORN. MARGARET WARWICK,
c. 158:95 (July) 1919.

s based on a study of necropsy findings. Little has been subject since 1885, at which time Sarah McNutt established between cerebral hemorrhage at birth and Little's disease. of necropsies reveals that intracranial hemorrhage is found in per cent. of cases. The location varies, the hemorrhage being rachnoidal, intracerebral, intraventricular, etc. The most fre- is over the cerebrum because the veins in this area are afforded m, and in rapid labor they have little chance to mold themselves ured by the over-riding of the parietal bones.

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DeLong, Philadelphia.

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Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

*Three Hundred and Seventy-Third Regular Meeting, held at the
Academy of Medicine, May 6, 1919*

WALTER TIMME, M.D., *President*

PRESENTATION OF CLINICAL MATERIAL. By DR. I. ABRAHAMSON.

Dr. Abrahamson presented six cases recovered from epidemic polioencephalitis and showed several photographs of the patients taken during the active stage of the disease. They all gave a history of epidemic influenza a short time before the development of the polioencephalitis.

CASE 1.—Present illness dated back five weeks. Patient showed Parkinson face, attitude and gait, typical tremor of paralysis agitans and cogwheel phenomenon. The condition had recently decidedly improved.

CASE 2.—The status showed a typical Parkinson face, attitude and gait, tremor mainly on intention, cogwheel phenomenon, rigidity of the extremities and bilateral facial weakness. There had been marked and constant improvement.

CASE 3.—The patient had had epidemic polioencephalitis one month ago. There was a lack of facial mobility.

CASE 4.—Four weeks ago the patient had encephalitis. He left the hospital almost well, but recently his head commenced to move involuntarily, breathing felt hampered, head and neck felt rigid, head bent forward to the right, and there was a beginning masklike face and attitude of a Parkinson. The Wassermann test of blood and spinal fluid was negative.

CASE 5.—Six weeks ago the patient had influenza. Since that time he had the following symptoms: dizziness, tinnitus, altered speech diplopia, drowsiness, mental confusion, confabulation, spasmodic cough, and hoarseness. There was present an external ophthalmoplegia, partial drowsiness, deviation of tongue to the right, weakness of right face and tremors. He had grown very stout since the onset of the illness. There was marked bulimia, polyuria, polydipsia, loss of ejaculatory power and impairment of taste. There had been a slow improvement.

CASE 6.—Initial symptoms which appeared in January, 1919, were headache, vertigo, gastric distress, weakness of extremities, especially the left, herpes labialis, fever; at first he was languid, then somnolent, eyes staring and pains in the extremities. There developed in the left leg a loss of control, then a tremor and then control of the left arm was lost and tremor developed. Following this his face became masklike, shoulder swing in walking was lost, he perspired freely, lost weight, there was difficulty in turning around quickly, retropulsion, marked slowness of all voluntary movements and twitching of right toes. On examination he presented all the signs of a typical Parkinson syndrome: masklike face, attitude and gait, tremor, rigidity and lack of associated movements. The course was progressive at first, but recently there had been steady improvement.

DR. C. C. BELING, Newark, presented a recovered case of epidemic encephalitis which showed the following sequelae: a general Parkinsonian attitude without muscular rigidity; fibrillary tremors of the tongue, face and lips; a high-pitched voice; drooling of saliva; slowness and monotony of speech; paralysis of accommodation of ocular muscles; and double facial palsy, more marked on the right. The patient had suffered from epidemic influenza and pneumonia in October, 1918, from which he apparently recovered. On March 4 he again had influenzal symptoms, followed suddenly by diplopia two days later. On March 8 he developed a spontaneous nystagmus on both sides, more marked on looking toward the right. On March 10 he became somnolent and lethargic and went into a stuporous, semiconscious condition which continued for about three weeks. During this period several lumbar punctures showed normal cerebrospinal fluid but under increased pressure. On March 29 the outstanding symptoms were a cerebellar attitude; bilateral facial paralysis, more marked on the left; increased knee reflexes, the right more than the left; tendency to hyperextension of big toes on stimulation of the soles, more marked in the right; slight clonic movements in the left ankle and a well-marked Oppenheim reflex on the right; numerous irregular movements of the eyeballs without any definite isolated nerve palsy; slight ectopia of the pupils to the inner sides; abdominal reflexes absent except for a slight left epigastric response; marked retardation of speech; general motility and anterograde amnesia for events of the previous two weeks.

CLINICAL EXPERIENCES WITH EPIDEMIC CENTRAL OR BASILAR ENCEPHALITIS. Presented by DR. B. SACHS.

This paper was based on his experience with about thirty cases at Mount Sinai Hospital and fourteen cases seen in consultation. There were only three fatalities in the former group which was not a high percentage, but out of the fourteen cases, five died with symptoms of marked bulbar involvement which made the prognosis in these cases ominous.

The term lethargic encephalitis was ill advised; it was not the encephalitis but the patient who was lethargic, and if one considered the predominance of central or basilar symptoms, a more appropriate name was not far to seek. The clinical symptoms were very striking; broadly speaking, after a brief period of drowsiness, headache, vertigo and general malaise, the patient passed into a state of lethargy associated with symptoms pointing to involvement of the cranial nerves. Meningeal symptoms were not obtrusive. The ptosis, ophthalmoplegia externa of the nuclear type, abducens paralysis, facial palsies, which were often double, cerebellar attitude of the head, difficulties of phonation and deglutition, fibrillary tremors of the tongue, double spastic paraplegias, all suggested an encephalitis that might involve the brain stem from the larger ganglions to the pons and medulla oblongata. In addition to these symptoms, the forced attitudes, catatoniac states, occasional impulsive laughter, masklike expression of countenance, even atrophies of the interossei muscles, led to the inference that the anatomic processes might involve the thalamus at one end and the cervical ganglionic cells at the other.

While awaiting definite proof of the nature of the virus, a study of which was being conducted by Strauss, Hirshfield and Loewe, one might lay stress on the fact that in many of the cases, and in the majority of those seen in consultation practice, there had been a distinct history of influenza preceding the onset of the lethargic disorder by several weeks. If this sequence was not a mere coincidence, this epidemic encephalitis bore a resemblance to the

postdiphtheritic palsies in its occurrence weeks after the initial infection. If this was a postinfluenzal infection, it was curious that previous epidemics of influenza had not been followed more frequently by similar disorders. The condition was so different from anything that had occurred before that it was impossible that it should not have been noted. The stupor was not the ordinary kind; the patient lay inert with closed eyes and expressionless face, but was apparently aware of what was going on about him and readily responded by nodding to questions that were put to him in a low tone of voice.

There was as yet no proof that the course of the disease was influenced by treatment, but it was a fact that the cases that recovered had received careful nursing and feeding. The treatment was eliminative and purely symptomatic in the absence of a specific remedy.

The prognosis of the disease was determined largely by the site of the lesion. All the fatal cases in this series had been bulbar forms. The patients had not succumbed to the toxicity of the disease but from the fact that the cardiac and respiratory centers had been the site of predilection.

Many authors had been inclined to note a resemblance between the poliomyelitis virus and the virus of epidemic encephalitis, but it was a point to remember that there was extreme toxicity and rapid development of the disease in fatal cases of poliomyelitis, while the fatal cases of epidemic encephalitis ran a course lasting from one to seven and eight weeks. The laboratory and experimental investigation would have to furnish conclusive evidence on this point.

REPORT OF THE COMMITTEE ON EPIDEMIC POLIOENCEPHALITIS (LETHARGIC ENCEPHALITIS).

Dr. I. Abrahamson, chairman of the committee, read this report which embodied the number of cases and seasonal incidence of all known epidemics of this disease up to the present time. An important difference between the epidemics in foreign countries and in the United States lay in the fact that the lymphocytosis so common here had been the exception abroad. Various names had been given to this disease: epidemic encephalitis, lethargic encephalitis, influenzal encephalitis, pontobulbar encephalitis, epidemic stupor, epidemic botulism, etc. The name polioencephalitis was preferable for three reasons: The brain was mainly affected in most cases, the nuclear involvement dominated the pathologic picture, and it was a companion disease to poliomyelitis. It was recommended that the term lethargic be dropped principally because lethargy characterized only a minority of the cases. It was pathologically and experimentally established that epidemic polioencephalitis was a distinct disease and one which until recently had escaped differentiation and recognition. Reasons were cited which would seem to indicate that there was no direct relationship between influenza and epidemic polioencephalitis. The differential diagnosis between epidemic poliomyelitis and epidemic polioencephalitis was equally demonstrable. As to the clinical manifestations of epidemic polioencephalitis, syndromes of all kinds could be established. This was an infectious encephalitis, the infection originating in the nasopharynx, proceeding by the lymph streams to the basilar cerebral vessels, and then spreading through the brain stem, ganglions and cortex and also involving the upper cord, the meninges and the nerves. Those portions of the brain immediately supplied by the cerebral and basilar arteries suffered first and most severely in the majority of cases.

DISCUSSION

DR. M. NEUSTAEDTER suggested that the best way to differentiate epidemic encephalitis from poliomyelitis would be to make the poliomyelitis neutralization test. A mixture of a 1:10 serum of a convalescent patient and true poliomyelitis virus injected into a monkey would certainly prove whether the condition were poliomyelitis or not. If the disease was poliomyelitis the serum ought to neutralize promptly the virus and the animal remain well. This suggestion was made in view of the report by the reader of the paper that encephalitis had been produced in monkeys by injecting them with a filtered suspension of the scrapings of the nasopharynx.

DR. SIMON ROTHENBERG thought that the most interesting phase of this disease was the variety of neurological syndromes that these cases presented. Although he had had eighteen or twenty cases, very few had shown identically the same picture. One simulated meningitis, another polioencephalitis with double facial palsy, a third was a cerebellar case, a fourth was of the dys-synergic cerebellar type of Hunt, a fifth was blind and showed marked coreiform movements, and finally, two other cases developed a picture of encephalitis during an attack of influenzal pneumonia. These last two cases were seen at the hospital where the first symptoms of the encephalitis were observed. In these cases the Pfeiffer bacilli were found in a culture from the throat, showing what was believed to be a connecting link between influenza and encephalitis.

DR. SMITH ELY JELLIFE recalled a family in which three patients were taken ill, apparently with influenza—one with influenzal pneumonia, who died, another with herpes and the third with a typical mesencephalitic affection. That incident had been duplicated three times in his experience and it would be interesting if it could be shown that another type of encephalitis had now been isolated which could be differentiated from an influenzal encephalitis. The first case of lethargic encephalitis he had seen was in 1890, and it would be fascinating if a new type of encephalitis might be differentiated from the others, pathologically differentiated, as this might enable still further the separation of types. From the year 1400 on, writers had accentuated the extreme variability of the different epidemic clinical manifestations which seemed to follow what were apparently clearly defined influenzal forms. It would be a great advance if one could get findings that would enable the separation of a new type producing mesencephalic manifestations.

DR. J. ARTHUR BOOTH said that during the influenza epidemic of 1889 he had seen two cases complicated by eye symptoms, in both of which there was an ophthalmoplegia externa, there being a bilateral ptosis and a paresis of the external recti muscles. There was an entire absence of undue somnolence and lethargy. In contrast with these he had seen three patients during the past winter in whom, with almost exactly the same eye conditions, there was the symptom of lethargy, and in one, a marked catatonic condition.

DR. WILLIAM M. LESZYNSKY referred to six patients that he had seen: One died at the end of two weeks; another was a physician whose particular symptom was twitching of the muscles in the extremities, but he made a complete recovery. Out of this small series only one died, all the others completely recovering. He had seen two or three cases which had been difficult to differentiate at first from the catatonic type of dementia praecox. In most of the cases gripe had occurred within a month previously.

DR. ELBERT M. SOMERS, Brooklyn, referred to the reports of forty cases called central neuritis in the 1908 and 1909 issues of the New York State Hospital's Bulletin. These cases had presented various stuporous states, muscular tension, jactitations and focal and mental symptoms. They appeared following grip, cancer, tuberculosis and infective exhaustive conditions. Thereafter, central neuritis was accepted as a cause of death by the health department of the state.

OBSERVATIONS ON GUNSHOT INJURIES OF THE HEAD. MAJOR

KARL WINFIELD NEY, M. C., U. S. Army, who was senior officer of the Neurosurgical Unit No. 1, A. E. F. in France, read this paper.

He emphasized the value of several procedures which experience in military cranial surgery had shown to be the cause of the immense reduction of mortality statistics. The necessity for early surgical intervention had been as pronounced in the cranial field as in others, and primary suture was the ideal treatment of all wounds. It had been found possible to effect this if all devitalized tissue could be excised before infection became established, and not only this, but certain wounds could be closed even when it was not possible to practice complete excision, and it was in this class that gunshot wounds of the brain had been placed. The surprising absence of sequelae—meningitis, brain abscess, hernia cerebri, brain fungus, etc., in these cases was most significant.

The two surgical principles of profound importance in this war, early and complete excision, gradually having found their places in the treatment of cranial injuries in so far as scalp and skull were concerned, the problem that then presented itself related to the removal of devitalized brain tissue. It was solved by Col. Harvey Cushing who combined the two principles just mentioned, and removed the disorganized brain substance by catheter suction and irrigation, completing the operation by primary closure of the dura and scalp.

In the endeavor to do a speedy operation, in the early part of the war, the procedure was often incomplete and frequently subdural adhesions were torn and the subarachnoid space opened to infection. The necessity for speed was due to the profound effects of general anesthesia on these septic patients, and realizing this the speaker became convinced that the same operative procedures were possible under local anesthesia. With the use of the same he succeeded in avoiding the shock associated with general anesthesia and was able to pursue a more deliberate operative technic without pain to, or interference from the patient, thereby insuring greater gentleness in manipulation, as well as a more careful toilet of the wound.

A 1 per cent. procain solution with a few drops of epinephrin chlorid was used and complete anesthesia was produced in less than ten minutes, which was a saving in time over general anesthesia. The infiltration of the scalp so reduced the bleeding that when the excision was made much time was saved in that only the larger vessels required clamping, and very often the field was bloodless. The hemostatic effect of the infiltration lasted always through the operation, or sufficiently long for clotting to take place in the constricted vessels, and in no case in this series did later hemorrhage occur. So satisfactory was local anesthesia in head operations that when there was a complication of other wounds the head operations were done under local anesthesia, and a general anesthesia given later for the debridement of other wounds. This applied not only to work at the front where complete operations

were done, but it proved to be just as successful at the base hospitals where operative procedures were done for brain abscess and other infective conditions associated with retained foreign bodies, cerebral hernia, fungus, etc. Another great advantage in the use of local anesthesia was the possibility of cooperation by the patient in asking him to blow his breath or cough; by thus increasing intracranial pressure it was possible to quickly remove the disorganized brain tissue, blood clots and often foreign bodies. In brain abscess it had proved most valuable, not only as to location but as to the area through which it might be approached.

Local anesthesia was *par excellence* the method of choice in cranial surgery. There was no pain in the bone, the dura was insensible to cutting though it would not stand traction or rough handling, and the manipulations of the brain itself never reached the threshold of consciousness. The operative technic, however, was of the greatest importance. After shaving the head and making as complete a neurological examination as possible, the following procedure was adhered to by Neurological Unit No. 1; complete excision of the scalp wound, avoiding contact with the lacerated edges; removal of the bone injury *en bloc*; evacuation of disorganized brain substance by having the patient blow against his closed lips or by coughing and also by catheter suction and irrigation; the detection of foreign bodies and bone fragments by catheter palpation and, after their removal, the instillation of dichloramin-T; and primary suture of dura and scalp. If the scalp defect was too large to permit suture without tension, the defect was covered by some plastic procedure. This was in many essentials the technic advocated and used by Colonel Cushing. In ventricular penetrations it was found possible, after removing the disorganized brain substance, to remove foreign bodies from the ventricles by direct inspection, using small retractors.

Observations had been made on a series of seventy-nine cases, thirteen of which were fractures with intact dura, the remaining cases representing all degrees of brain injury associated with lesions of venous sinuses, ventricular penetrations, and combined lesions with frontal sinus or mastoid complications. Nine of the thirteen fractures with intact dura were complicated with either extradural or intradural hemorrhage, producing compression symptoms of varying degree. The total mortality was represented by five deaths. Judging from reports on hand and from many cases personally examined by Major Ney, he did not believe that the late complications would be many. In the examination of about 200 cases not a single one of abscess or cerebral hernia had been observed in any having had the complete early operation.

DISCUSSION

DR. ALFRED S. TAYLOR expressed his appreciation of the privilege of listening to Dr. Ney's splendid paper in which there were two things that impressed him particularly: the very great interest of the intrinsic material of the article, and the complete lucidity of the presentation of a complicated and difficult subject. The application of cranial surgery as performed in the army to civil life was plain, and it was also clear that one should apply local anesthesia more than one had been accustomed to do.

DR. HAROLD NEUHOF said that Major Ney had presented the best results that had been obtained in gunshot wounds of the brain of the American Expeditionary Forces. The earlier mortality figures in dural penetration were about 50 per cent. With the improvement in technic, as advocated chiefly by Cushing, this was reduced to about 30 per cent. and most of the surgeons

were satisfied to have it at that low figure. Major Ney's was therefore a remarkable achievement. The technic he described was one that was followed, with a few variations, by all the teams under Cushing's command with parallel results. The principle had been to permit dural defects, even if large, to remain as such. Dr. Neuhof himself believed that some form of dural repair was indicated and, when the tear was too large, he employed transplantation of fascia. Fascia lata was employed and entered into the dural defects. The results were satisfactory, both immediately and as later reported. Dr. Neuhof stated his belief that local anesthesia for operations on the head not only proved the method of choice for war wounds but would similarly prove the anesthesia of choice in head operations in civil life. Since his return he had done several operations on the head under local anesthesia, among them a bilateral suboccipital craniotomy, as well as an osteoplastic flap in the parietal region, with results that encouraged its further use in head surgery.

COL. EDWIN BEER added a few words from his personal observations confirming Major Ney's statements. He declared that there was no doubt that these results were the best attained, but whether credit was due to Major Ney's own skill than to the use of local anesthesia there was some question. Dr. DuMartell of Paris told the speaker that he did all his civil skull and brain surgery under local anesthesia and had even removed a cerebellar pontine tumor, which was a difficult procedure. The most remarkable thing, proved by Ney and others, was the discovery that the brain was able to stand so much contamination without disastrous effect. One could not possibly get absolute sterility in these war wounds, and yet the dura could be closed as well as one could close up the knee joint, showing that the vital processes could be trusted to cope with the residue of infectious material which could not be removed.

DR. SACHS considered that the two most impressive points about this address were: First, the observation that local anesthesia acted as a hemostatic of the scalp; he had often thought that the large number of deaths in children following cranial surgery was due to loss of blood. Secondly, he was interested to learn that the surgeon's finger was not in future to be brought in contact with the brain substance and hereafter only the catheter would penetrate the depths of the brain. It would also be a great aid to the recognition of the presence of abscess on the operating table if the patient himself under local anesthesia could assist by exerting intracranial pressure through blowing out his cheeks, indicating the exact site and thus doing away with so much of the indiscriminate puncturing of the brain tissue that had been necessary in times gone by. It had been a delightful privilege to listen to this clear exposition of such valuable experiences and remarkable results as Major Ney had attained.

Book Reviews

THE AUTONOMIC FUNCTIONS AND THE PERSONALITY. By EDWARD J. KEMPF, M.D. Nervous and Mental Disease Monograph Series, No. 28, 1918.

A new viewpoint will be given to many readers by this monograph. In it psychiatry is found looking toward physiology for help, but modestly suggesting that it has knowledge of its own to give in return.

The first part of the book is given over to establishing a great reflex arc with the whole autonomic apparatus as its afferent arm and the cerebrospinal nervous system as a proficient arm. Experimental work (Cannon) has shown gastric contractions are concomitant with hunger pangs. Using this and other physiologic facts (Sherrington, Crile) as a basis, the author claims that emotion comes into existence only as peripheral autonomic reactions become aroused. Cannon's statement that hunger "may take imperious control of human actions" is given as the key to the dynamic functions of the personality, taking for granted that all autonomic cravings have the same physiologic function as hunger. The efferent side of the great arc of the cerebrospinal system is to be considered only as an agent developed and used by autonomic apparatus to get this or that subject in order to satisfy (neutralize) an effective craving. Hunger drives the brain and its striated muscles to get food. Thus "the mind" is brought down from its high throne and scattered among a lot of lowly organs.

The functioning of this great reflex arc as it deals with external stimuli in differing accidental associations (the conditioned reflex of Bechterew) determines the acquired traits of the personality and the symptoms of mental disease. Into his own psychobiologic terms the author goes on to translate the *libido* and other psycho-analytical conceptions, the higher emotions, memory, consciousness and life in all its aspects.

In a study of tonus in Part 2 the book best deserves the attention of the psychiatrist. After a consideration of "postural tonus" (Sherrington) and its dependence on the continuing activity of many reflexes, it is argued that it is foolish to consider that there is no emotional state when the individual shows no perturbation in his behavior. "An effective status continually exists." It is noticed that postural tonus varies with the affect as when one drops a lightly held razor on hearing surprising news. An affective craving which cannot find satisfaction through the cerebrospinal system is seen as stored in a heightened postural tension of some viscus. Attention is called to the apparently unchanged muscle tonus of catatonia and of agitated melancholias.

In a sense the important things described in this book are true whether the contentions in it are later upheld or not. The writer has described at least one side of a fundamental situation, an achievement worth while.

NEUROLOGICAL CLINICS. Edited by JOSEPH COLLINS, M.D. Paul Hoeber, New York.

This book is made up of 271 pages. It consists of a series of case reports with the discussion chiefly concerned in the diagnosis and differential diagnosis. The cases recorded are for the most part of cerebral or spinal origin and are

derived from the first division of the Neurological Institute. Collins is aided in his work by the various members of his staff.

The cases reported are practically all of organic nature. They are well presented, and the discussion which is incident to each case is very often illuminating.

The reviewer was made "tres triste" by a remark on the first page made by the editor. Collins states very naively that the diagnosis of the brain is largely a matter of guesswork. Then he proceeds to diagnose his case very skilfully and with an acumen that belies the foregoing remark.

CESARE LOMBROSO. *Storia della vita e delle opere narrata dalla figlia.* Turin, 1915.

In the volume before us Signora Ferrero has told, with much sympathy and natural appreciation, but with good taste and restraint, the story of her father's long struggle for recognition against prejudice and a curious opposition.

Cesare Lombroso was born of a well-to-do and eminent Spanish-Hebrew family in Verona in 1835, but soon after his birth the family met with financial reverses and his means became limited. His earlier education was subject to Austrian tyranny. He was obliged to go to a school under Jesuit control, and under such surveillance that a comrade once reported to the police his possession of a copy of Lucretius. An ardent and precocious student, fond of literature, Lombroso, after the reopening of the universities, entered the school of medicine at Pavia, at the age of 17, and from thence, three years later, he went to Vienna. His mind was filled with a desire to benefit his fellows by study of the treatment of some of their more distressing ills, and to do for his countrymen what was being done for the people of Vienna, especially in the treatment of mental diseases. To that end he began a study of cretinism in Lombardy, making important contributions to the etiology and treatment of that affection. On his return to Verona the political situation led him to enter the medical service of the Piedmontese army, and after the close of the war he was transferred to the division hospital at Pavia, where he again began to turn his attention to mental diseases and to study especially the relation between the insane and the criminal. In 1864 he left the army to devote himself more especially to the study of mental disease, but returned for the war of 1866. After finally leaving the army, he took up the study of pellagra, at that time extremely prevalent in Italy. Lombroso claimed that pellagra was due to a toxin formed in the perisperm of spoiled maize, and that it could be successfully combated by arsenic and by a regulation of the diet, excluding maize. His studies were presented in 1870 to the Lombard Institute, which offered a prize for a memoir on pellagra of definite benefit to society. The Institute, while highly commending Lombroso's work, failed to award him the prize, which his work apparently merited. In 1871 he was appointed to take charge of a hospital for the insane at Pesaro; but, owing to the lack of students and the absence of facilities for teaching, he soon returned to Pavia. In the meantime, Lussana, professor of physiology at Pavia, had attacked Lombroso's doctrines in regard to pellagra. In 1874 he accepted the chair of legal medicine, not including mental diseases, at Turin. Soon after, the Lombard Institute again took up his claims as to the origin of pellagra, and a most acrimonious attack followed. It is difficult to understand the animus which led to the attack on Lombroso's doctrines, and the apparent unfairness which actuated his opponents. But the story, as told by Signora Ferrero, shows a disregard of evidence strange in scientific men,

although she does not fully make clear the reason for the virulence with which he was attacked. Lombroso's experiments were confirmed by other observers outside of Italy and in time gained acceptance even in Italy itself, and it is only of late years that further research has thrown doubt on some of his theories. In 1875 he went to Turin as professor extraordinarius in legal medicine instead of in psychiatry, with scanty facilities for his anthropologic and criminal museum, without the clinical facilities of a hospital for the insane, and with merely such clinical facilities as were furnished by the prison of Turin, of which he was made physician. The following year he published his *Trattato antropologico sperimentale dell' Uomo delinquente*, which while meeting with marked success elsewhere in Italy, in Turin was received with much the same disfavor as his work on pellagra. The following year he published a new edition of his *Genio e Follia*.

In the meantime he had married and had several children. For several years his life at Turin was a hard one. With scanty means, inadequate facilities, unsatisfactory clinical advantages, and lack of recognition, he struggled on, in hope of ultimate triumph, declining a better position at Reggio-Emilia because there he would have no teaching opportunities. A second edition of his great work, now called simply *L'Uomo Delinquente*, was called for and attracted attention all over the world. In the early eighties he started the *Archivio di Psichiatria*. His views on pellagra began to meet with more general acceptance and when the first International Congress of Criminal Anthropology was held Lombroso was accorded wider recognition outside of Italy. In spite of this foreign recognition, the new penal code of Italy, put forward in 1888, failed to adopt the chief tenets of his teaching, and the second Congress of Criminal Anthropology, held in Paris in 1889, showed considerable opposition, Manouvrier claiming that although there was truth in Lombroso's system, the system as a whole was false. In 1891, however, Lombroso was appointed to the full professorship of clinical psychiatry in Turin, to succeed Morselli. He was also made inspector of asylums. A short time later, in conjunction with Tamburini, he examined Eusapia Palladino and failed to expose her trickeries. In consequence of his investigations, he openly espoused spiritualism, writing a treatise in its defense. In 1899 a law was passed dealing with pellagra and adopting, in the main, Lombroso's theories. From that time on he led an active life at Turin, writing a great deal, upholding his own views, and receiving many honors as the pioneer of criminal anthropology, although the controversies over his views continued until his death in 1909.

Writing as a devoted disciple of her father's doctrines and as a zealous advocate of his views, Signora Ferrero fails fully to make clear the motives for the opposition to Lombroso or set forth the arguments against his teachings. Consequently, it is difficult for a foreigner to understand the motives back of the opposition and to comprehend the apparent unfairness of many of the polemics against him, so that in reading this story of Lombroso's struggles and the contests that embittered his life almost to its close, the natural query is whether her filial partisanship has not led her to give a somewhat prejudiced picture. Like all pioneers, Lombroso was an enthusiast and had the imaginative temperament of the poet rather than the cold judgment of the scientist, although the latter was not lacking. Hence we find in *L'Uomo Delinquente* not only the exact and arid measurements of the criminal but also many speculative theories as to the identity of the *criminale nato*. In his earlier years Lombroso was a disciple of homeopathy, in later life

THE UNITED STATES OF AMERICA
DO hereby certify that the within and foregoing is a true and correct copy of the original as the same appears in the records of the Department of the Interior, Bureau of Land Management, at Washington, D. C.
In testimony whereof, the Chief of the Bureau has hereunto set his hand and the seal of the Department at Washington, D. C., this 1st day of January, 1900.

The undersigned Special Agent in Charge will pay the
value of the land and improvements thereon to the
owner or his heirs, assigns, or assigns, and will
execute all necessary documents to carry out the purpose
of this act.

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THERAPY OF NEUROSYPHILIS JUDGED BY ARSENIC PENETRATION OF MENINGES

METHODS OF TREATING NEUROSYPHILIS *

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AND

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SAN FRANCISCO

Of the methods of neurosyphilitic treatment, the more commonly used are the following:

(a) Intensive antisyphilitic medication; this consists of arsphenamin given intravenously, mercury and the iodids with such hygienic and supportive treatment as may be indicated for the individual case. In favor of this treatment is its simplicity and its relative freedom from complications. Arsphenamin given intravenously results invariably in arsenic penetration into the spinal fluid according to Benedict,¹ and in only 30 per cent. of the cases according to Reiger and Solomon.² The clinical results obtained vary widely; some cases are very successful, others less satisfactory, and some types are absolutely resistant to this form of medication.

(b) Drainage of the spinal fluid following previous intravenous injection of arsphenamin has been advocated. It is argued that drainage of spinal fluid increases the amount of arsenic in the spinal fluid following the intravenous injection. This method is simple and has but little unfavorable reaction. The clinical results occasionally are good, but perhaps not better than those treated by the simple intensive method.

(c) The intradural methods of Swift and Ellis, Ogilvie and Byrnes attempt to still further increase the concentration of a spirochetacidal substance in the spinal fluid by introducing into the subarachnoid space blood serum reenforced with arsenic or mercury. Against these methods have been urged the technical difficulties in giving the treat-

* From the Neurological Clinic of Stanford University Medical School.

1. Benedict, cited by Sachs: *Am. J. M. Sc.* **148**:693, 1914.

2. Reiger, J. B., and Solomon, H. C.: The Circulation of Arsenic in the Cerebrospinal Fluid, *J. A. M. A.* **71**:15 (July 6) 1918.

ment, painful reaction to the patient and complications that have followed especially when the serum was too highly fortified in the Ogilvie and Byrnes modification. The clinical results in the hands of many investigators have been especially satisfactory in cases resistant to other forms of treatment.

Up to the present time there has been considerable difficulty in estimating the relative values of the above methods. Each method has its advocates who report series of cases in which the majority have improved under the particular treatment employed.

VALUE OF METHODS TESTED BY PENETRATION

It has even been denied that the presence of an antispirethetal substance in the spinal fluid was of advantage to the patient. While this point is not entirely clear, there is evidence tending to show that certain types of meningitic lesions are amenable to local treatment. This is particularly true of Flexner's intraspinal treatment of epidemic cerebrospinal meningitis. The lesions of neurosyphilis are so diffuse in location, kind and intensity that therapy directed to the meninges alone would benefit only a limited number of patients. Clinical results have shown that the parenchymatous lesions of paresis are least benefited by the introduction of spirochetacidal substance into the spinal fluid. Meningeal lesions are the most benefited. Inasmuch as we are unable to state the exact moment at which a given case of neurosyphilis passes from a meningeal to a parenchymatous state, therapy directed to the meninges would seem to have a considerable field of usefulness.

The concentration of spirochetacidal substance in the spinal fluid necessary to get clinical results is another mooted point. Whether reinforcing the serum with arsenic or mercury as done in the Ogilvie and Byrnes method gives us an increased therapeutic effect sufficient to offset the increased meningeal irritation, is again not clear. Swift³ himself believes that the high concentration is unnecessary. He bases the success of the intradural methods more on the spirochetacidal action of a serum with a low arsenic concentration—the presence of syphilitic antibodies in the serum and local action of an irritating effect produced by serum and chronic inflammatory process. On the basis of experiments on dogs and man, he differs with Sicard⁴ in believing that the irritation of the meninges was unable to cause a let down of the barrier between the blood and spinal fluid.

3. Swift, Homer F.: Intraspinal Therapy in Syphilis of the Central Nervous System, *J. A. M. A.* **65**:209 (July 17) 1915. Stillman and Swift: Cited by Swift in same article.

4. Sicard, J. A.: *Bull. et mém Soc. méd d. hôp. de Par.* **36**:681, 1913.

OUTLINE OF PRESENT INVESTIGATION

The following experiments were undertaken to find out if the normal penetration of arsenic into the spinal fluid could be increased by an irritation of the meninges such as must occur in all of the intradural treatments. It seemed important to reinvestigate this point as the studies of Flexner and Amos⁵ on poliomyelitis had demonstrated a definite let down in the barrier between the blood and spinal fluid that could be caused by a variety of irritants.

CHEMICAL ANALYSIS

Quantitative estimations of the arsenic penetration in the meninges have been made on approximately 100 spinal fluids after the various methods of treatment. The analytic method used in detail is as follows: The sample of cerebrospinal fluid is placed in a 200 c.c. round bottom flask. Five c.c. of arsenic-free strong nitric acid and 3 c.c. of pure sulphuric acid are added. The contents are boiled slowly until the brown fumes cease, then concentrated until the first appearance of the white sulphur trioxid. The sulphuric acid should be nearly colorless; if not, a few more cubic centimeters of nitric acid are added and the digestion repeated. To the cooled acid 5 c.c. of water are added and boiled again until the first appearance of white fumes. Heating should be avoided after sulphur trioxid appears. About 15 c.c. of water are added and allowed to cool to room temperature. The fluid is then transferred to a 30 c.c. bottle and the volume made up to 25 c.c.

Through the cork of the bottle is a glass tube, the dimensions of the lower part of which are 1 cm. by 10 cm., and the upper part drawn out to 0.4 cm. by 6 cm. In the lower part of this tube near the constriction, glass wool moistened with a solution of lead acetate is placed with dry lead acetate paper below. In the constricted part of the tube is inserted a 0.3 cm. by 6 cm. strip of mercuric chlorid paper (filter paper soaked in 5 per cent. mercuric chlorid, dried and placed in stoppered tube). Ten gm. of arsenic free granulated zinc are just covered with 5 per cent. pure copper sulphate. This is washed free from the copper sulphate and added to the 25 c.c. of solution in the bottle; the cork and upright glass tube are immediately inserted. A rapid stream of hydrogen is evolved for some time. In about twenty minutes 1 c.c. of 5 per cent. copper sulphate is added to the bottle; this will again increase the rate of evolution of gas. In forty minutes from the addition of the zinc compare the intensity and extent of the color on the mercuric chlorid strip with standard strips of same sample

5. Flexner, S.: Neutralizing Substances in Poliomyelitis, *J. Exper. Med.* 25:499 (April) 1917; Amos, H. L.: *Ibid.*

TABLE 1.—GROUP A—CONTROLS

Serial No.	No. of History	Diagnosis	Cells before Treatment	Cell Reaction	Amount of Fluid Withdrawn, C.c.	Amount of Fluid Examined for Arsenic, C.c.	Total Arsenic in Mg.	Arsenic in Micro-milligrams per C.c.
1	66105	Cerebrospinal syphilis	2	5	25	15	0	0
2	63911	Syphilis	0	0	20	10	0	0
3	65976	Tabes	6	6	20	10	0.0005	0.05
4	60673	Cerebrospinal syphilis	60	4	22	12	0	0
5	13181-13	Tabes	6	0	25	15	0	0
6	52727	Tabes	6	1	22	12	0	0
7	50861	Cerebrospinal syphilis	3	1	25	15	0.0004	0.03
8	29145	Cerebrospinal syphilis	51	8	25	15	0	0
9	65976	Tabes	6	6	25	15	0	0
10	64706	Cerebrospinal syphilis	30	48	25	15	0.0010	0.07
11	44000	Syphilis	2	10	30	20	0.0008	0.04
12	60698	Syphilis	0	1	30	20	0	0
13	50981	Cerebrospinal syphilis	40	4	25	15	0.0015	0.10
14	54706	Cerebrospinal syphilis	30	23	30	20	0	0
15	50981	Cerebrospinal syphilis	40	4	30	20	0.0040	0.20
16	51750	Tabes	1	2	30	20	0.0005	0.04
17	55404	Syphilis	0	0	20	10	0.0003	0.03
18	60698	Syphilis	0	38	30	20	0	0
19	60698	Syphilis	0	9	25	15	0	0
20	60698	Syphilis	0	8	25	15	0.0006	0.03
21	63727	Tabes (burnt out)	2	2	20	10	0	0
22	63727	Tabes (burnt out)	2	0	20	10	0	0
23	60442	Cerebrospinal syphilis	10	10	25	15	0	0
24	60442	Cerebrospinal syphilis	10	34	20	10	0	0
25	60442	Cerebrospinal syphilis	10	2	25	15	0	0
26	54706	Cerebrospinal syphilis	30	5	25	15	0	0
27	60797	Tabes	28	5	25	15	0	0
28	49866	Syphilis	1	1	25	15	0.0010	0.07
29	50861	Cerebrospinal syphilis	3	1	25	15	0	0
30	63930	Syphilis	1	1	40	30	0	0
31	30688	Cerebrospinal syphilis	9	9	20	10	0.0004	0.04
32	65473	Tabes	5	3	20	10	0.0010	0.10
33	49866	Syphilis	1	1	22	12	0.0006	0.05
34	50981	Cerebrospinal syphilis	3	1	25	15	0.0010	0.07
35	49866	Syphilis	1	1	10	10	0.0040	0.40
36	43525	Cerebrospinal syphilis	47	10	30	20	0	0
37	44000	Syphilis	2	10	20	10	0.0010	0.10
38	66361	Cerebrospinal syphilis	10	10	20	10	0	0
39	66766	Cerebrospinal syphilis	445	1560	25	15	0	0
40	44469	Tabes	5	5	20	10	0	0
41	66105	Cerebrospinal syphilis	2	10	20	10	0.0007	0.07
42	44469	Tabes	5	2	30	20	0.0010	0.05
43	66105	Cerebrospinal syphilis	2	5	20	10	0	0
44	51750	Tabes	1	0	25	15	0.0006	0.04

of paper colored by known amounts of arsenic produced in the same apparatus under the same condition as the unknown. In this way 0.1 mmg. (1 micromilligram = 0.001 milligram) of arsenic can be detected. Up to 1 mmg. the results are probably correct to 0.2 mmg. Between 1 and 10 mmg., the error is probably not greater than 0.5 mmg.

TABLE 2.—GROUP B

Serial No.	No. of History	Diagnosis	Cells before Treatment	Cell Reaction	Amount of Fluid Withdrawn, C.c.		Amount of Fluid Examined for Arsenic, C.c.		Total Arsenic in Mg.		Arsenic in Micro-milligrams per C.c.	
					First Drainage	Second Drainage	First Drainage	Second Drainage	First Drainage	Second Drainage	First Drainage	Second Drainage
1	54293	Tabes.....	11	—	50	30	40	20	0	0	0	0
2	50661	Cerebrospinal syphilis	2	—	45	35	35	25	0	0	0	0
3	44878	Tabes.....	20	—	45	30	85	20	0	0	0	0
4	28386	Cerebrospinal syphilis	30	—	55	35	45	25	0.0004	0.0007	0.01	0.03
5	68229	Tabes.....	23	—	25	20	15	10	0	0	0	0
6	18841	Cerebrospinal syphilis	17	—	45	30	85	20	0	0	0	0
7	64706	Cerebrospinal syphilis	30	—	55	45	45	35	0.0003	0	0.01	0
8	60698	Syphilis.....	0	—	30	30	20	20	0	0	0	0
9	58556	Cerebrospinal syphilis	48	—	20	25	20	15	0	0	0	0
10	29145	Parasitis.....	4	—	35	30	25	20	0	0	0	0
11	29145	Cerebrospinal syphilis	51	—	35	35	25	25	0.0004	0.0004	0.02	0.02
12	62047	Tabes.....	4	—	40	25	30	15	0	0.0003	0	0.03
13	51750	Tabes.....	1	—	25	30	15	20	0.0006	0	0.04	0
14	50861	Cerebrospinal syphilis	2	—	40	30	30	25	0	0	0	0
15	49866	Syphilis.....	1	—	25	30	15	20	0	0	0	0
16	52727	Tabes.....	1	—	50	35	40	25	0.0003	0	0.007	0
17	54293	Tabes.....	14	—	50	30	40	20	0	0	0	0
18	70143	Cerebrospinal syphilis	14	—	?	?	?	20	0	0.0003	0	0.012
19	70143	Cerebrospinal syphilis	14	—	?	35	?	25	0	0	0	0
20	69649	Cerebrospinal syphilis	4	—	50	25	40	15	0	0	0	0
21	69649	Cerebrospinal syphilis	4	—	30	30	20	20	0.0016	0	0.08	0

TABLE 3.—GROUP C

Serial No.	No. of History	Diagnosis	Cells before Treatment	Cell Reaction	Amount of Fluid Withdrawn, C.c.	Amount of Fluid Examined for Arsenic, C.c.	Total Arsenic in Mg.	Arsenic in Micro-milligrams per C.c.
1	46608	Tabes	6	—	30	20	0	0
2	70659	Cerebrospinal syphilis	14	—	25	15	0	0
3	15108	Tabes	451	—	25	15	0	0
4	70649	Parasitis	14	—	25	15	0	0
5	58556	Cerebrospinal syphilis	45	—	30	20	0	0

RESULTS OBTAINED

GROUP A consists of forty-four cases in which spinal drainage was performed one hour following simple intravenous injection of 0.6 gm. arsphenamin. We chose the interval of one hour, as Reiger and Solomon found that arsenic concentration in the spinal fluid attained its maximum at this time. Forty-three per cent. of these cases gave

positive tests for arsenic, averaging 0.036 mmg. of arsenic per cubic centimeter of spinal fluid.

GROUP B consists of twenty-one cases in which intravenous injection of arsphenamin was followed in less than an hour by a complete drainage of the spinal fluid. One hour after the first drainage a second lumbar puncture was done to determine whether complete drainage tended to increase the amount of arsenic penetration. In 29 per cent. of the cases arsenic penetrated in half an hour; in only 19 per

TABLE 4.—GROUP D

Serial No.	No. of History	Diagnosis	Cells before Treatment	Cell Reaction	Temperature Reaction	Amt. of Fluid Withdrawn, C.c.	Amt. of Fluid Examined for Arsenic in C.c.	Total Arsenic in Mg.	Arsenic in Micro-milligrams per C.c.
1	44469	Tabes.....	5	6	101.0	20	10	0.008	0.30
2	27530	Tabes.....	4	2,300	100.5	25	15	0.002	0.13
3	27530	Tabes.....	4	385	102.0	13	3	0.0065	0.17
4	2093	Paresis.....	26	125	99.8	25	15	0.001	0.07
5	18841	Cerebrospinal syphilis	17	126	99.8	25	15	0.0004	0.03
6	65473	Tabes.....	5	2,040	100.5	25	15	0.002	0.13
7	46808	Tabes.....	10	1,660	101.0	25	15	0.002	0.13
8	66105	Cerebrospinal syphilis	2	108	99.0	22	12	0.004	0.33
9	58556	Cerebrospinal syphilis	48	80	98.2	25	15	0.004	0.27
10	58556	Cerebrospinal syphilis	48	24	98.6	30	20	0.001	0.05
11	56530	Tabes.....	5	191	99.2	17	7	0.003	0.43
12	56530	Tabes.....	5	456	99.5	30	20	0.006	0.40
13	50931	Cerebrospinal syphilis	40	3,289	101.0	20	10	0.0005	0.05
14	60442	Cerebrospinal syphilis	10	98.0	25	15	0.001	0.07
15	60442	Cerebrospinal syphilis	10	710	100.8	25	15	0.0005	0.03
16	60498	Syphilis.....	0	103	99.6	30	20	0.0007	0.04
17	60767	Tabes.....	5	765	99.5	30	20	0.0005	0.03
18	60673	Cerebrospinal syphilis	6	2,980	99.0	35	25	0.001	0.04
19	52727	Tabes.....	1	61	98.8	35	25	0.0006	0.02
20	62047	Tabes.....	4	75	100.4	30	20	0.001	0.05
21	62047	Tabes.....	4	19	99.2	25	15	0	0
22	60442	Cerebrospinal syphilis	10	99.4	25	15	0.0008	0.05
23	14361	Cerebrospinal syphilis	23	1,320	101.0	20	10	0.0005	0.03
24	64818	Cerebrospinal syphilis?	3	1,136	100.0	25	15	0.001	0.07
25	65547	Tabes (burnt out).....	5	4,200	101.2	25	15	0.0015	0.10
26	61725	Cerebrospinal syphilis	48	2,000	99.5	25	15	0	0
27	49690	Cerebrospinal syphilis	44	470	101.5	20	10	0.0008	0.06
28	63727	Tabes (burnt out).....	2	693	101.0	30	20	0.003	0.15
29	68925	Cerebrospinal syphilis	60	525	100.5	30	20	0.0004	0.02
30	68925	Cerebrospinal syphilis	60	750	100.0	30	20	0.002	0.10
31	46808	Tabes.....	10	1,600	101.4	35	25	0.0015	0.06
32	41067	Cerebrospinal syphilis	1	39	100.0	16	6	0.002	0.33
33	68925	Cerebrospinal syphilis	126	1,350	100.5	25	15	0	0
34	65714	Cerebrospinal syphilis	19	19	98.8	45	35	0	0
35	30882	Tabes.....	1	1,380	101.0	35	25	0.0010	0.04
36	20145	Cerebrospinal syphilis	11	99	99.6	35	25	0.0003	0.012
37	70849	Cerebrospinal syphilis	15	298	102.0	25	15	0.0008	0.05
38	70143	Cerebrospinal syphilis	17	127	101.0	30	20	0.0008	0.04
39	69649	Cerebrospinal syphilis	4	320	99.0	20	10	0.0005	0.05
40	70849	Paresis.....	15	298	102.0	25	15	0.0008	0.05

cent. of the cases was arsenic found on the second drainage. The average amount of arsenic penetrating in the first drainage was 0.008 mmg.; in the second drainage was 0.0044 mmg. Allowing for the fact that this group contains a smaller number of cases than Group A, we can conclude that the number of penetrations is certainly not greater than that obtained by simple intravenous injection.

GROUP C consists of five cases in which a complete drainage was made an hour before the intravenous injection of arsphenamin and

this showed no arsenic penetration an hour after the injection. Apparently, placing the drainage before instead of after the intravenous injection has no advantages.

GROUP D consists of forty cases in which the patient's own serum was injected into the subdural space followed in from six to eight hours by an intravenous injection of arsphenamin. One hour after the injection a lumbar puncture was made. Ninety-two per cent. of these cases gave a positive test for arsenic in the spinal fluid. Quantitatively, they averaged 0.103 mmg. per cubic centimeter. The meningeal irritation resulting from the serum injection ranged from a reaction 100 to 2,300 cells per cubic millimeter.

APPLICATION TO TREATMENT

It would seem that the foregoing results could be applied to the treatment of neurosyphilitics in the following way: (1) Every case should have the benefit of intensive intravenous medication until we are certain that the case belongs among those with impermeable membranes. (2) Our experiments would suggest that for the cases resistant to ordinary therapy, in order to obtain the maximum concentration of arsenic in the spinal fluid, the patient's own blood serum should be injected into the subarachnoid space six hours before the arsphenamin is given intravenously. This serum may be injected as in the Swift-Ellis technic, or, if necessary, arsenic or mercury may be added according to the Ogilvie or Byrnes technic. This procedure is not more complicated than either of the intradural methods, and has the definite advantage of allowing arsenic to pass from the blood to the spinal fluid in greatly increased concentration. Clinically, we have carried this process through in eighty cases. These cases were chosen because we had treated them previously by the ordinary intensive antisyphilitic treatments with meager results.

SUMMARY

Irritation of the meninges by intradural injection of the patient's own serum caused a cellular reaction ranging from 100 to 2,300 cells per cubic millimeter of spinal fluid.

2. Simple intravenous injection of 0.6 gm. arsphenamin resulted in a positive test for arsenic in the spinal fluid in 43 per cent. of the cases.

3. Complete drainage of the spinal fluid did not increase the number of arsenic penetrations.

4. Intravenous injection of arsphenamin six hours after meningeal irritation gave 92 per cent. penetrations and compared with the controls, gave three times as strong an average concentration of arsenic.⁶

6. In addition to the references given, the following may be of interest: Barbat, J. H.: Permeability of the Meninges to Arsenic in Paresis and Tabes, *J. A. M. A.* 70:147 (Jan. 19) 1918.

LOCOMOTOR DISTURBANCES IN DISEASE OF THE CEREBELLUM

A GRAPHIC STUDY

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CHICAGO

In a recent communication¹ I reported the results obtained by graphically recording the gait of an animal after two different types of experimental lesions of the cerebellum. The records obtained indicated changes, characteristic of the type of lesion, which could not be detected by ordinary observation. This led me to believe that the graphic study of locomotion, which Marey truthfully designated "the microscope of movement," might be useful in the study of cerebellar disease in men: It might indicate not only the presence, but also the location of a lesion. For this method I had to consider:

First. The type of locomotion should be one in which all the limbs are in action. Even in the ordinary progression of man, there is simultaneous activity of all the extremities, each arm swinging forward with the advance of the contralateral leg.² But this activity of the arms is rudimentary, inconstant and irregular, and could hardly be utilized for cerebellar localization. Still, the action of the arms should be studied because it is generally accepted that the *crus primum* of lower animals³ (corresponding to the *lobus quadrangularis* and *lobuli semilunares* in the cerebellum of man) dominates the homolateral anterior limb. Failure to study movements of the upper limbs would leave us in the dark as to whether such disorder in the lower limbs is due to a lesion in the leg area of the cerebellum, or is merely secondary to a lesion in the arm area.

Second. The phase of locomotion studied should be that of extension. This phase is undeniably an expression of neuromuscular activity, and depends on integrity of the cerebellum. The flexion stage, on the

1. Meyers, I. Leon: The Cerebellar Gait. A Pedographic Study, *J. Nerv. & Ment. Dis.* **48**:14, 1919.

2. Graham, Brown: Note on the Movements of Progression in Men, *J. Physiol.* **14**:17, 1912; Pettigrew, J. Bell: *Design in Nature*, New York, Longmans, Greene & Co. **3**:1120, 1908; Thompson, H. Campbell: Associated Movements, *Brain* **26**:518, 1903.

3. Meyers, I. Leon: Cerebellar Localization. An Experimental Study by a New Method, *J. A. M. A.* **67**:1745 (Dec. 9) 1916; Van Rynbeck, G.: *Erg. d. Physiologie* **12**:536, Asher-Spiro's, Wiesbaden, 1912.

other hand, may be brought about entirely by the mechanical laws governing a compound pendulum.⁴

These two requirements could best be met, in my judgment, by recording the extension phase in a person ascending a stairway with a convenient railing on either side of him. In this type of progression both upper as well as lower limbs are almost always in action, the tendency being to help the legs by pressure of the hands on the railing. Generally, the contralateral arm and leg act together, especially if the subject's attention be distracted from his extremities. In consciously innervating the lower limbs a normal person may not use the arms at all. But this interference with the experiment is much less apt to be present if the person ascending the stairway is suffering from weakness or unsteadiness in the legs, because then he seeks to compensate by use of the arms.

In studying the simultaneous action of an upper and its contralateral lower limb we bear in mind that there is no strict parallelism in the action of the two. The action of the lower limb, to use the simile employed by Borelli, is of the same character as that of the boatman's oars which advance the boat by propelling the resistant water toward the stern. By extension this limb exerts pressure on the ground, which being an unyielding medium, reacts on the body, propelling it in the opposite direction, i. e., forward. The action of the upper limb is like that of the boathook which causes the boat to come to the shore by being fastened to a stationary object and being pulled on. However, the action of either limb is brought about by neuromuscular activity, and either limb employs the extensor muscles (as discussed farther on). I see no objection, therefore, to considering the movements as similar and comparable.

THE APPARATUS FOR CARRYING OUT THE EXPERIMENTS

Lower Limbs.—Two pieces of heavy linoleum fitting the sole of the foot are sewed together. In the lower are cut two fenestra, one for the heel, one for the ball of the foot, and into each is fitted an air bulb (1 and 2) easily made from a piece of rubber tubing. From each air bulb leads a piece of slender rubber tubing (3 and 3) to a glass T tube (4), the soles being grooved for its passage. This double sole with air bulbs is then encased in heavy cloth (6) and the whole securely bound to the foot with tapes. A rubber tube 20 feet long (5) connects the stem of the glass T tube (4) with a Marey's tambour.

4. Weber, Wilhelm and Eduard: *Mechanik d. Menschlichen Gehwerkzeuge*, Goettingen, 1836; Wilhelm Weber's *Werke*, Verlag von Julius Springer, Berlin, 1894, Vol. 6; Duchenne, G. B.: *Physiologie des Mouvements*, 1867, p. 410.

Upper Limbs.—An air chamber (3) is made of two circular steel plates about $1\frac{1}{2}$ inches in diameter (4), and a piece of rubber tubing of sufficient caliber to be hermetically fastened to the periphery of each. Into what is to be the upper plate is soldered a steel communication tube from which leads a rubber tube (5) connecting this air

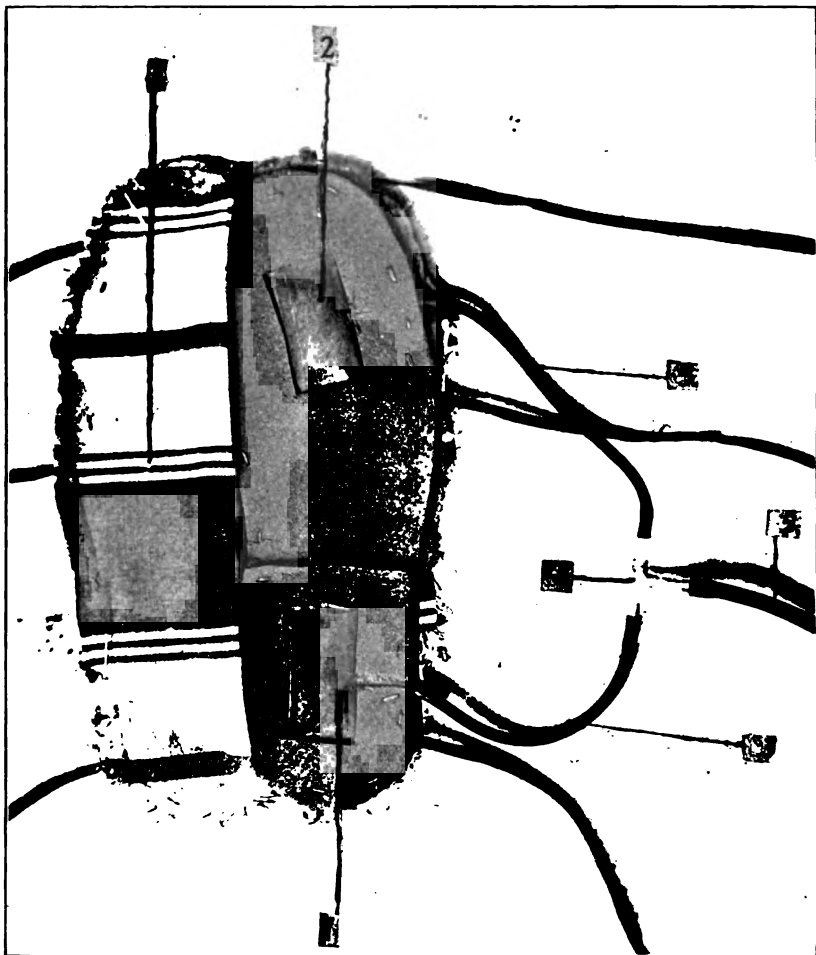


Fig. 1.—Apparatus for recording the movements of the lower limbs.

chamber with a Marey's tambour. This chamber is held firmly on the back of the hand by means of a rigid steel bar (2) fastened to its upper disk. The other end of the bar is soldered to a steel half-wristlet (1) embracing about the dorsal half of the lower forearm, where it is securely fastened in place.

Each of the four appliances on hand and foot having been fastened in place and connected with its respective tambour, and each tambour having been connected with a proper revolving drum of a kymograph, we are ready to make records.

THE NORMAL GAIT

The subject is instructed to ascend the stairway, with attention diverted from the movement of his limbs. In starting he would better have one hand rest on the railing, the other free. The rapidity with which the drum is allowed to revolve should vary in accordance with the pace of the individual. He should ascend at least eight or ten steps. The gait records thus obtained, while subject to some irregu-

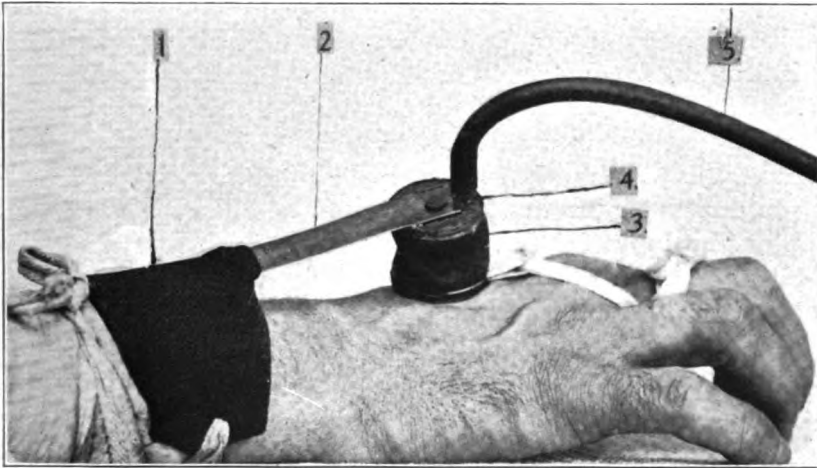


Fig. 2.—Apparatus for recording the movements of the upper limb.

larities, due to peculiarities of gait, excessive consciousness of the movements and to mechanical imperfections, shows definite characteristics, some constant, some frequent and others only occasional. It can be identified as that of a normal person.

Figure 3 shows such a record. It is typical of a large number obtained from three normal individuals (all right handed). It shows: First, that in the human being, as in the lower animals, when progression is carried out by all four extremities, there is a synchronous diagonalism, one upper and its contralateral lower limb acting simultaneously to produce the same effect, in this instance to advance the body upward and forward.

Secondly, that in this type of progression there is a period in each step when both feet are on the ground. This is also shown in the

tracings of Marey,⁵ who, in his studies of a man walking upstairs, recorded the movements of the lower limbs only. The period appears to be of much longer duration in my records than in his, perhaps due to difference in inclination of the stairway or in height of the steps. This period is absent in ordinary locomotion on level ground.

Thirdly, that while the upper limb and its contralateral lower limb differ in their modes of action, they initiate and complete their action by four different movements (indicated by *a, b, c, d*, for the upper,

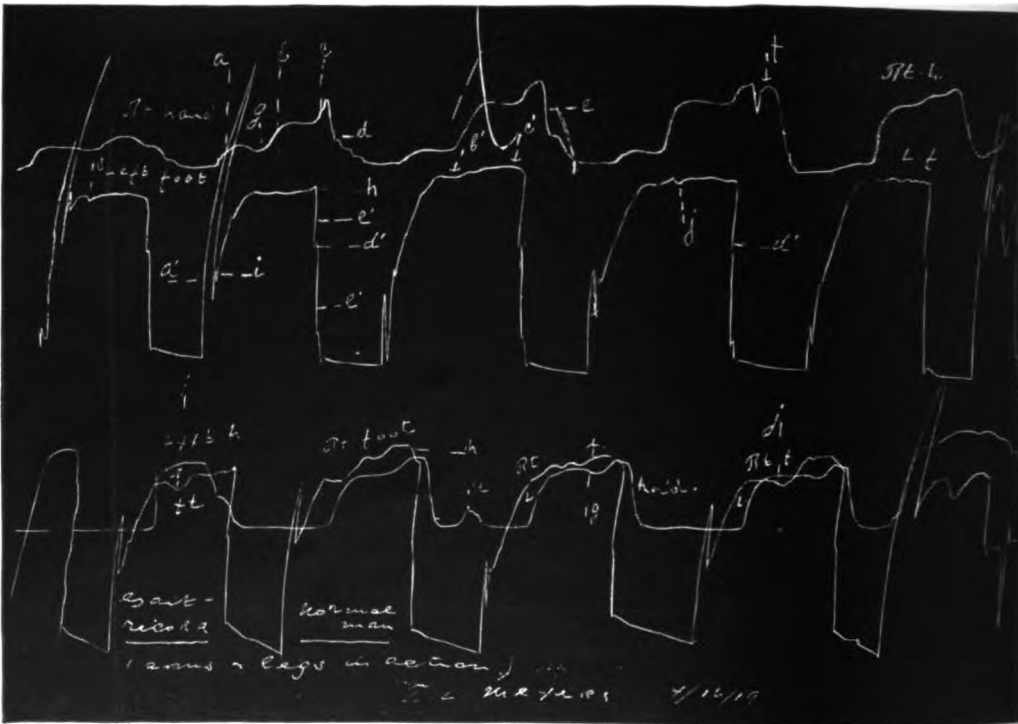


Fig. 3.—Gait record of normal man.

and *a', b', c', d'* for the lower limb). These four movements in the upper limb are as follows: During the stage *a* the advanced hand gently seizes the railing. This contraction of the flexors digitorum is associated with contraction of the extensors of the wrist, the moderating synergy of Duchenne.⁶ The slight extension of the wrist forces air from the chamber on the dorsum of the hand into the tambour, thus producing the upstroke of its recording lever.

5. Marey, E. J.: *Animal Mechanism*, New York, D. Appleton & Co., 1874, p. 127.

6. Duchenne, G. B.: *Physiologie des Mouvements*, 1867, p. 161.

This is followed by stage *b* (in the right-handed individuals stages *a* and *b* in the left upper limb are frequently concurrent so that one stage instead of these two, is recorded), in which the fist is clenched about the railing and contraction of the extensors of the wrist is intensified as described by Duchenne,⁶ Beevor and Horsley,⁷ and Hering.⁸ Thus more air is forced into the tambour, resulting in a further rise in the curve. Stage *b* represents the period during which the person by a pulling effect on the railing helps to elevate his body to the step above.

Stage *c* is the period during which both feet are on the ground. The extension of the wrist now reaches its maximum, the palm presses on the railing and compression of the air chamber is at its greatest, translated by the greatest elevation of the curve. Stage *c* is terminated by a momentary relaxation in all the muscles of the upper limb, pressure on the air chamber is released, and the lever falls (*e*). During this relaxation the hand may rest passively on the railing, or it may be pendant. In the latter instance, frequently there is a very slight rise in the curve (*f*) which is due to slight extension of the wrist associated with freeing the finger from the railing. The relaxation following *c* is very brief as it is interrupted by another movement of extension, comparatively slight in extent (stage *d*) which is associated with the advance of the hand in its effort to seize the railing again with the inauguration of another step. To carry out this movement the hand not only has to advance forward and upward, but has to be elevated above the railing. This is accomplished by extension at the wrist. When the hand reaches a point above the railing, it is allowed to fall, becoming relaxed again.

Another feature shown by the record is that strong contraction of a group of muscles frequently is preceded by slight contraction of the antagonists. Thus immediately preceding the vigorous extension of the wrist in stage *b* or *c* frequently there is a slight momentary fall in the curve (*g*), which seems to indicate contraction of the flexors. This phenomenon has been described by Beevor⁹ as characteristic of hysterical hemiplegia. My records indicate that it occurs to a slight degree in the normal person.

The four movements by the lower limb in executing the step synchronously with its contralateral upper, correspond with the four

7. Beevor, Charles E., and Horsley, Victor: Phil. Tr. Royal Soc., London **178**:160, 1887.

8. Hering, E. A.: Arch. f. d. ges. Physiol. **70**:559, 1898.

9. Beevor, Charles: The Croonian Lectures, London, Adlard & Son, p. 55, 1904.

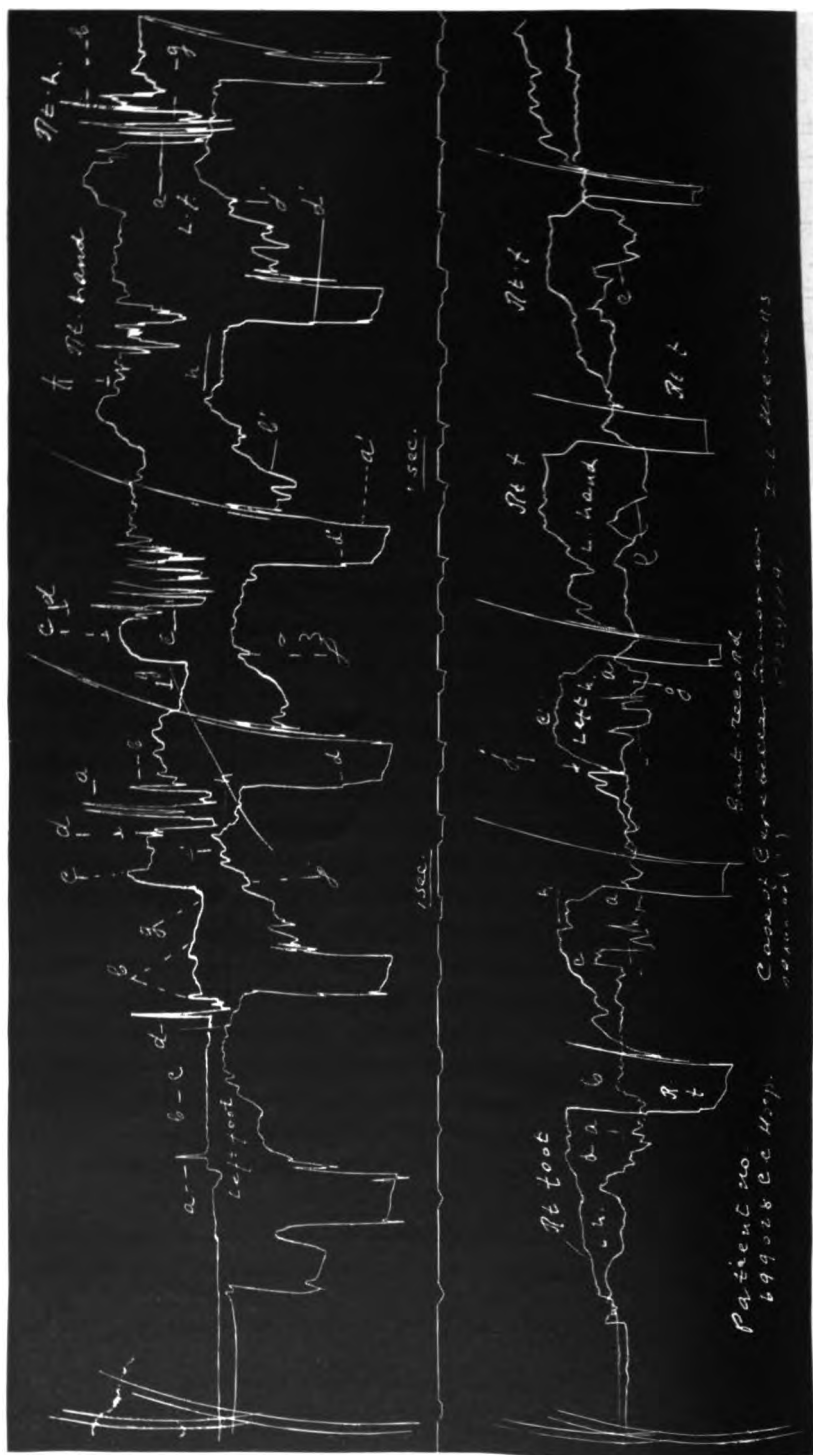


Fig. 4.—Gait record of patient with cerebellar disease.

stages; described by Philippson,¹⁰ in the hind limb of the dog in the ordinary trot. The record shows first, the movement indicated by *a'*. During this movement the limb is extended at ankle and knee, but slightly flexed at the hip (extension phase i of Philippson) and the heel falls to the ground. This movement (*a'*), according to the brothers Weber, Pettigrew and others, may be mechanical, due to gravity and totally independent of neuromuscular activity. Thus the heel bulb is compressed, causing the upstroke on the record. The height of this rise varies, depending on the rapidity with which the foot falls to the ground. If the foot comes down quickly and strikes the ground with considerable force the lever rises far beyond the point it would reach through gradual pressure. The lever then falls (*i*), the extent varying with the extent of its original rise. (It is possible that here, too, a contraction of the antagonists may play a rôle.)

Stage *b'*, which immediately follows, is produced by a movement of extension at the hip, as the result of which pressure is exerted on both toe and heel bulbs. The foot during this stage becomes dorsiflexed on the leg (extension stage ii of Philippson). This stage is followed by stage *c'*, in which there is complete extension of the limb at hip, knee and ankle (extension stage iii of Philippson). Pressure by the ball of the foot is greatly intensified; amounting, according to Carlet,¹¹ to about one-fifth in excess of the body weight. This movement of extension reaches its maximum at the peak of the curve, labeled *h*, whereby the body is elevated to the level of the step ahead. Immediately preceding stage *c'* there is a brief and slight depression in the curve (*j*). This depression, I believe, results from the person exerting the pressure in two stages (*b'* and *c'*). As the weight is transferred from the bulb at the heel to the bulb at the ball of the foot the former is released more rapidly than the latter is compressed. This would diminish pressure in the tambour, causing the recording lever to fall. Still, it is possible that here, too, contraction of the antagonists may cause this slight fall.

Stage *c'* is terminated by relaxation of all the extensors, resulting in the fall *e'*, which is terminated before reaching the abscissa by another movement of extension, rise *d'*. This movement is not shown in the tracings of the gait in walking upstairs made by Marey,⁵ but a repetition of Marey's experiments as well as of my own work have convinced me that the movement indicated by *d'* is one of the most constant characteristics of this type of locomotion. In fact, the tracing from a hemiplegic can at once be identified by this phase alone. It

10 Philippson, M.: *L'autonomie et la centralisation dans le système nerveux des animaux*, Bruxelles, p. 16, 1905.

11. Carlet, G.: *Annale des Sciences Naturelles*, Tome 16, S. 5, 1872.

(*d'*) corresponds to the moment when the foot is about to leave the ground and advance. During movement this pressure is exerted by the toes only, the ball of the foot being off the ground. (At this moment the body is supported by the other foot which in its turn has taken the step ahead.) The purpose of the movement at *d'* is probably to propel the limb itself upward and forward in the same manner as the latter, by its extension during *b'* and *c'*, propels the body in this direction—*d'* thus inaugurates the advance of the limb. The pressure at the toes causes the sole and air bulb to be bent slightly upward, thus producing slight tension recorded at *d'*. In hemiplegia, as I hope to discuss more fully in a subsequent paper, owing to the failure of the paralyzed limb to propel the body properly, the normal limb, which is in contact with the ground by the toes, assumes the burden and the extension of the limb at *d'* is greatly prolonged. Normally, immediately following stage *d'* the limb advances to the next step, i. e., to reach the second stair step above. During this phase, of course, there is no record made by this method.

My records also show that the movements on the right are generally much more vigorous than on the left; probably because all my subjects were right-handed.

THE CEREBELLAR GAIT

In the following case I was able to determine the presence of, and localize a cerebellar lesion by, the above described method when other clinical methods had failed. I should add that I knew nothing of the patient except that he was supposed to be suffering from some inflammatory process in the cerebellum. I believe that this method will be of practical clinical value and at least merits further study, although more extended investigation with improved apparatus may modify the tracings and the conclusions to be drawn therefrom.

AUTHOR'S CASE

History of the Patient.—F., aged 23, entered the Cook County Hospital, Chicago, Feb. 15, 1919, on the service of Dr. G. B. Hassin.

On Nov. 27, 1916, having been perfectly well, he awakened with severe frontal headache and dizziness and soon vomited. On rising he was very unsteady and noticed marked oscillation of the eyes. On the same day he had a discharge from one ear. A few months later his head began to tremble continually. He remained at home for four months and was then in the Buffalo General Hospital for over a year. He has somewhat improved; the headaches, dizziness, vomiting and sensation of bad odor have disappeared, but he still has tremor and unsteadiness involving head, trunk and all limbs. He also has an intermittent discharge from the right ear.

History of Symptoms.—Headache was frontal, at first severe, lasted about two years and suddenly ceased. Vomiting was at first severe, gradually grew less and disappeared with the headaches. Vertigo was bad at first; objects

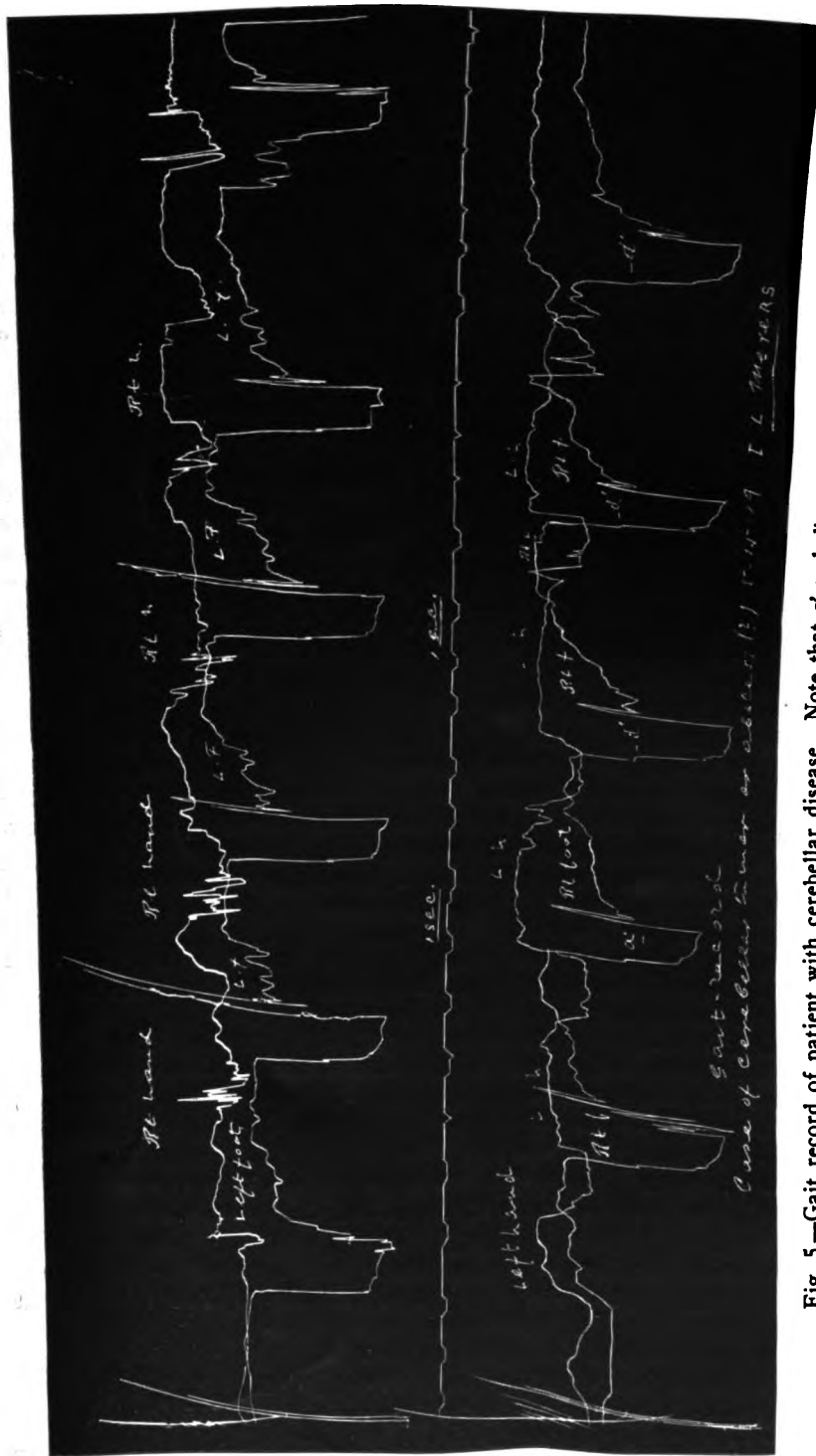


Fig. 5.—Gait record of patient with cerebellar disease. Note that α' and α'' are unaffected in the lower limbs.

moved to and fro and from left to right; the body swayed, more to the right. Nystagmus has gradually grown less. Vision has always been good. Parosmia appeared with onset and persisted for twenty-three months. There have been aural discharges at intervals since onset, the last occurring about three months ago. Unsteadiness on feet has improved. Tremor of the head and body appeared with onset, was worse four months later, then less.

Examination.—The general condition was good. Abdominal and thoracic organs and genito-urinary system were negative. No paralysis, no paresis, nor sensory changes found. No atrophy; neither hypertonus nor hypotonus. The deep reflexes were rather brisk but equal on the two sides; superficial normal. The pupils were equal and normal. There was slight tremor of the tongue.

There was a general tremor, most marked in head and arms, equal on the two sides. There was also a coarse, general incoordination of the "muscle group" or extremity type. When sitting, the body and head swayed from side to side.

The patient had difficulty in rising from a chair, and could not stand or walk unaided. He had a tendency to fall in any direction, but more often to the right. The whole condition was of the "cerebellar type." Hypermetria and adiadokocinesis were equal on the two sides; asynergia not marked. Speech is slow, labored, broken.

Nystagmus is present, quick component to the right. He past points with the right hand about 4 inches to the right and with the left hand 4 inches to the left.

The otologic department reported:

1. Neither labyrinth appeared over-sensitive and the spontaneous nystagmus was not exclusively of vestibular origin.
2. The right labyrinth showed evidence of degeneration which may readily be explained on the basis of a running ear and the physical findings in the ear.
3. Positive evidence of nerve deafness on the right side.

Otherwise cranial nerves were normal. No abnormal position of head.

Laboratory examination of urine, blood and spinal fluid was negative. Roentgen-ray examination of head was negative.

Diagnosis: Infectious involvement of right middle ear, mastoid and vestibule with extension to the cerebellum. Nature and location of cerebellar lesion was undetermined; operation advised.

Dr. George W. Boot operated on April 5, 1919, exposing the cerebellum, mostly on the right, and probed the right lateral lobe. Nothing abnormal was found and the patient remained *status quo*. Dr. Hassin then referred the patient to me and he was investigated by the method indicated in the foregoing. The tracings showed (Figs. 4 and 5):

1. During such progression there was marked tremor of the four limbs.
2. The lower limbs, in general, carried out the movements of a normal person.
3. Aside from the tremor, the only noteworthy abnormality in the movement of the lower limbs was that the relaxation immediately following the maximum extension at h, was delayed; the contraction of the extensors had a tendency to continue.
4. The changes in tracing of the right upper limb were as follows:
 - a. The movements of extension in stage a, following relaxation in stage c, were exaggerated to a remarkable degree. Stage a, was not, as in the normal

person, a maintained movement but was broken up by violent tremor into its component contractions.

b. Stage *a* set in prematurely, at a time when the contralateral lower limb, owing to its delayed relaxation, was still on the ground. This accords with my experimental results in dogs.

c. There was some exaggeration of the extension which inaugurated the stage *b*.

d. The flexion of the wrist at *g*, which was frequently absent in the gait of a normal person, was marked and rather prolonged in this type of pathologic gait.

e. The initial movement at *c*, following the slight relaxation preceding it at *g*, was also, like this movement in *b*, exaggerated, but not excessively so.

f. Stage *d* was exaggerated to a very remarkable extent.

In short, the gait records of the patient showed marked hyperactivity in the musculature of the right upper limb. Such hyperactivity was, as I have shown,¹² the essential, all-important manifestation of a cerebellar lesion. As before noted, the lobus quadrangularis and lobula probably represent the homolateral upper extremity. The head and trunk are generally conceded to be dominated by the vermis. It therefore appeared to me almost certain that the seat of cerebellar involvement in this patient was in the right lobus quadrangularis at or near the vermis. Accordingly, I advised a second operation to explore this area, which Dr. Boot was kind enough to perform on May 30, 1919. He found the lobus quadrangularis and superior lobus semilunaris near the vermis firmly adherent to the tentorium by numerous vascular adhesions. These were broken up and the profuse bleeding that followed was controlled by pressure. A flat gutta percha drain was inserted between the cerebellum and tentorium, and the wound closed.

Evidently the patient was suffering from a localized meningo-encephalitis as a result of extension of infection from his right ear.

The patient made an uneventful recovery. At the time of the present writing, four weeks after the operation, he shows marked improvement in his cerebellar symptoms. The tremor and ataxia have greatly diminished, especially in his head and upper limbs. As the cortex of the cerebellum at the site of the lesion probably has been severely damaged, complete recovery is not to be expected.

SUMMARY

The locomotor disturbances in disease of the cerebellum are shown, by the graphic records, to be the expression of two abnormalities in the action of the voluntary motor system: First, a hyperactivity of the muscles, as a result of which the movements are greatly exaggerated. The hyperactivity of the extensors is shown by the records to be especially marked when their contraction follows a period of complete relaxation. It is thus much more striking during the stages *a* and *d* which follow complete relaxation at *e*, than in stage *b* or *c* which follow the partial relaxation at *g*. In previous publications on the

12. Meyers, I. Leon: A Galvanometric Study of the Cerebellar Function, J. A. M. A. 65:1348, 1915.

cerebellum¹³ I have advanced the theory that the essential function of the cerebellum is to inhibit; to regulate the activity of the motor cortex and of the tonus centers, probably located in the medulla. This theory seems to be supported by the graphic records here reported.

Second, a tremor disturbing the smoothness of movement. A movement is brought about by the fusion of a number of successive muscular contractions. In this tremor each contraction is separated from the other by a period of relaxation. If we assume, as before suggested, that each contraction of a muscle is preceded by a slight contraction of the antagonists, it is possible that the dissolution of a movement, intended to be maintained, into its component contractions (tremor), as is shown so conspicuously during the stage *a*, is not due to alternate contraction and relaxation of the muscles in action, but to a hyperactivity of the antagonists which *precedes* each contraction of the acting group. If this theory is correct, the tremor is but a variant of the excessive activity of the muscles resulting from a lesion of the cerebellum—it is a manifestation of excessive activity in the antagonists which precedes the intentional contraction by the active group.

This work has been carried out at the Cook County Psychopathic Hospital, and I am greatly indebted to Dr. Clarence A. Neymann, superintendent of this hospital, and to Dr. G. B. Hassin, in charge of the laboratories there, for furnishing me so generously all facilities for the experiments.

25 East Washington Street.

13. Meyers, I. L.: (Footnotes 3 and 12).

THE ACTION OF CERTAIN DRUGS ON BRAIN CIRCULATION IN MAN *

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The pharmacologic response of the brain vessels in man is a question presenting more than ordinary difficulty to experimental study, particularly under normal, nonoperative conditions. The only work of any significance in the latter connection is that of Shepard,¹ and even here the matter of specific drug effect is but incidentally dealt with—only one group, that of the nitrites, having been definitely reported. It was deemed distinctly fortunate, therefore, when the presentation of a suitable subject rendered further investigation possible.

REPORT OF CASE

The subject R. K., aged 40, a male patient at the State Psychopathic Hospital, had undergone double subtemporal decompression for the relief of increased intracranial pressure, presumably of tumor origin. The first decompression had been performed nine months, and the second, eight months prior to the initiation of this study, the patient, having by that time, fully recovered from all operative reaction. There developed residually, however, at the decompression sites, two large, pulsating cerebral hernias, each about the size and shape of a lemon. Mentally, the patient, though showing a certain deterioration, was fully capable of satisfactory cooperation.

METHOD OF PROCEDURE

Specifically, the procedure was as follows: The subject was required to lie at full length on his left side, with his face flat to the pillow, thus furnishing a level and stable support for the presenting right hernial mass, and extreme care was taken to maintain this position throughout each observation period. The site of the presenting mass was shaved and encircled by a bed of putty into which was set, so as to enclose the mass completely, an ordinary glass funnel of suitable size. To the tip of this was affixed a length of rubber

* From the laboratories of the Department of Pharmacology, University of Michigan, and the State Psychopathic Hospital, Ann Arbor, Mich.

1. Shepard: *The Circulation and Sleep*, New York, Macmillan Co., 1914, p. 80 et seq.

tubing, provided with a regulating T-arm, and terminating in a delicate recording-tambour of the Meister type, which was so placed as to record on a moving drum. It was found easily possible in this manner to secure a perfectly satisfactory pulse tracing. As a preliminary precaution, in order to rule out possible skin and muscle effects, duplicate appliances were affixed over the forehead, jaw, and thorax; these, however, yielded no demonstrable pulsation or other change.

RESULTS

The first drug investigated was amyl nitrite, administered by inhalation. The response was immediate and frank. There was noted

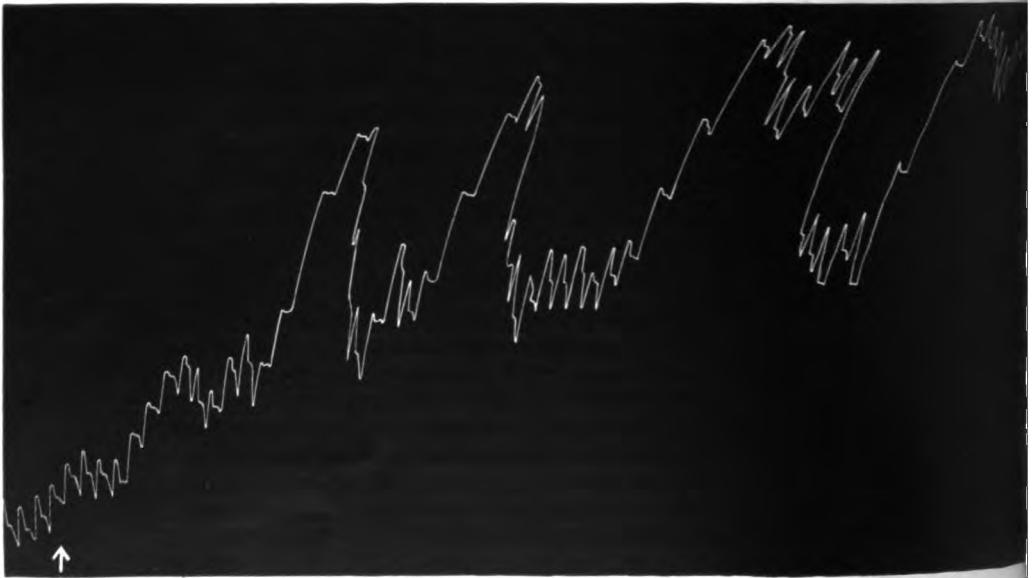


Fig. 1.—Pulse tracing showing effect of amyl nitrite.

(Fig. 1) a distinct upward movement of the lever indicative of increased volume in the part under observation, i. e., vasodilation. This was attended by a distinct increase in the pulse rate, the beat showing characteristic diastolic pulsation. These findings were distinctly in corroboration of Shepard's.¹ They were also in agreement with Hirschfelder's² results, as determined through observation on the retinal and pial vessels of cats and rabbits.

The second drug studied was epinephrin, administered intravenously, very slowly and in minute, diluted dosage until effects were observed. The response to this drug, also, at every trial, seemed

2. Hirschfelder: J. Pharmacol. & Exper. Therap. 6:597, 1914-1915.

quite definite. There was noted first (Fig. 2) a slight but distinct constriction, soon followed by a marked dilation, the reaction being characterized throughout by an increased pulse rate and amplitude. There was no demonstrable "leukoreaction" or blanching of the face, as reported by Milian.³ It is conceivable that these results may be explained on the basis of a primary specific brain vessel constriction which is soon overcome, mechanically, by the massive reflux of blood from other more powerfully contracting centers, such as the splanchnic and limb areas. These observations seem of particular interest in view

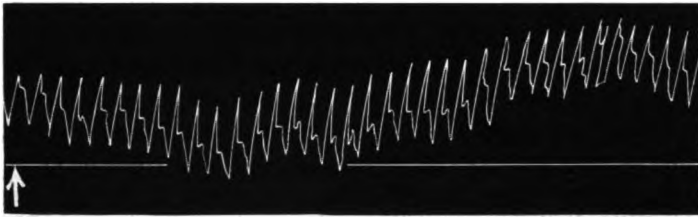


Fig. 2.—Pulse tracing showing effect of epinephrin.

of the prevailing differences of opinion in regard to the exact response of the brain circulation to epinephrin. The only available findings approaching those of the present study have been reported by Kahn⁴ based on observation of the retinal vessels of cats and rabbits.

The drug next investigated was caffeine—administered, intravenously, as caffeine—sodium benzoate, each dose containing 3.75

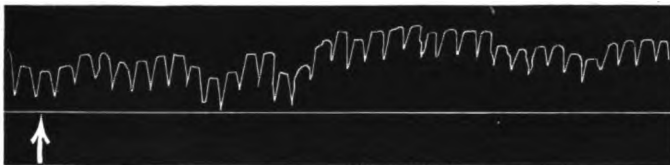


Fig. 3.—Pulse tracing showing effect of pituitary extract.

grains of the alkaloid. The findings in this connection were negative—no change in rate or volume being demonstrable. Such a response is readily conceivable in the normal animal when one considers the opposite tendencies excited by caffeine, to wit: bulbar vasomotor stimulation as against local vasodilation and vagus excitation as opposed to direct stimulation of the heart muscle. Still it is possible that, with greater dosage, a more definite reaction might have been secured. The

3. Milian, G.: Administration of Epinephrin, *Paris méd.* **8**:81 (Feb. 2) 1918.

4. Kahn: *Centralbl. f. Physiol.* **18**:153, 1904.

results seem to be at variance with findings by Berezin⁵ and Hirschfelder.² Both these investigators, the former working with the perfused brain in pikes and rabbits, and the latter on the pia and retina of cats and rabbits, have reported a dilation. Roy and Sherrington,⁶ however, in their volumetric studies on trephined dogs, claim an initial increase in volume followed by a decrease which, in turn, is succeeded by another rise.

The last drug exhibited was pituitary extract (pituitrin, Parke, Davis & Co.). This also was administered intravenously very slowly and in minute diluted dosage until effects were noted. In this connection there was found (Fig. 3), characteristically, a distinct though slight rise in volume, unaccompanied by any definite change in pulse rate or amplitude. It is probable that the dilation here may have the same basis as indicated in the case of epinephrin, i. e., representing a passive distention in accommodation to the generalized systemic constriction induced by the drug. It seems worthy of note that, after pituitary extract, there was observable what might be regarded as a marked "leukoreaction"—the subject evidencing a distinct greenish pallor accompanied by a moderately profuse perspiration, though complaining of no actual bodily discomfort.

SUMMARY

On the basis of plethysmographic observation on the brain circulation in man under normal, *in vivo*, condition, it seems that:

1. Amyl nitrite causes a marked dilation of the brain vessels.
2. Epinephrin induces a primary constriction of the brain vessels, which is followed by a marked dilation.
3. Caffein produces no demonstrable change in the dosage employed.
4. Pituitary extract is followed by a dilation of the brain vessels, accompanied by a distinct "leukoreaction."

5. Berezin: *Russk. Vrach* **15**:513, 1916.

6. Roy and Sherrington: *J. Physiol.* **11**:85, 1890.

A STUDY OF THE INVALID REACTION

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Perhaps no class of patients inflicts so much strain on the time, patience and medical wisdom of the general practitioner and specialist in every branch of the profession as persons with hypochondriac complaints who drift with the tide of every day farther and farther out into a condition of invalidism. On the other hand, it must be admitted with equal regret that no class of patients suffers more at the hands of the general practitioner and specialist than do these unfortunate members of society. If one cares to burrow into the mass of literature that has accumulated about the subject, and to separate case histories from the luxuriance of terminology and classification by which they have become overgrown, he can satisfy himself that the above statements are not exaggerated. The helplessness of efforts to serve this large body of ailing human beings seems due to a persistence in our thinking of what Adolf Meyer has called "the medically useless contrast of mental and physical."¹

With the introduction of "neurasthenia" by Van Deusen half a century ago, herculean efforts were made to hunt down a physical basis both for its somatic symptoms referable to every system in the body, and for its "psychic" manifestations which were elastic enough to include every reaction except well-formulated types of hysteria, organic brain disease and the major psychoses. The results were set down years later by Cowles² of Boston in the Shattuck Lecture for 1891 wherein it was claimed that the neurasthenic suffers from an exhaustion of the nervous system in which the nutrition of the nerve cells is primarily at fault. As a consequence, there is increased irritability and fatigability on the nerves per se. Meanwhile, Weir Mitchell³ had systematized a "cure" designed to minister to the faulty nutrition with rest, fat and blood.

But there were those in the profession who found it hard to believe that the "psychic" manifestations of the severer cases could be

1. Meyer, Adolf: Objective Psychology or Psychobiology with Subordination of the Medically Useless Contrast of Mental and Physical, J. A. M. A. **65**:860 (Sept. 4) 1915.

2. Cowles, Edward: Neurasthenia and Its Mental Symptoms, Boston M. & S. J. **125**:73 (July 23) 1891.

3. Mitchell, Weir: Fat and Blood, Philadelphia, J. B. Lippincott Co., 1884.

accounted for on the basis of actual nerve exhaustion. Among these early objectors was Janet,⁴ who undertook to give a psychologic explanation for these psychopathologic facts. According to him, there is in the main a lowering of mental energy in these individuals that side-tracks productive activity into indecisiveness, obsessions, phobias, tics, etc., that are amenable to an arrangement in five groups, depending on the degree of disturbance presented by the patient. These cases Janet withdrew from the main body of neurasthenia, and called them psychasthenia. The term is still applied by the internist⁵ to those cases of neurasthenia in which the "psychic" side predominates; it is applied by the psychiatrist to psychopathologic conditions associated with obsessions, fears and states of tension; and it is recognized by Freud as practically synonymous with his *Zwangsneurose* (compulsion neurosis) which he includes in the psychoneuroses. Another dissenter from the original doctrine of neurasthenia as a fatigue neurosis is Freud, who divides neuroses into physioneuroses, or *Aktualneurosen*, and psychoneuroses. Both are due to difficulties in the sexual life; the one explicable on more or less of an organic basis, or at least on the ground of a disorder of the functions; the other only on a psychologic basis. The physioneuroses consist of "true neurasthenia" with all the somatic symptoms described by Van Deusen and later by Beard. According to Freud, this is due wholly to masturbation, which, as an inadequate substitute for normal coitus throws undue strain on the energy of the individual. In fact, Freud often refers to this syndrome as "masturbation neurasthenia."⁶ In contradistinction to the latter in mechanism, but also a physioneurosis, is an anxiety state characterized by a constant feeling of vague dread punctuated at times by panic attacks associated with palpitation, dyspnea, choking sensations, perspiration, etc. From Freud's standpoint, these symptoms represent accumulations of somatic sexual excitement aroused by such frustaneous attempts at satisfaction as coitus interruptus, etc. Denied an outlet they find expression in states of tension and anxiety which Freud has called the anxiety neurosis.

Throughout this nosological confusion one sees the passion to isolate, and to divide and subdivide syndromes according to whether they can be explained as physical or mental in origin. The consensus of opinion seems to hold that every somatic complaint must have a physical basis in actual nerve tissue deficit from intoxications of either fatigue or sexual substances. As to the "psychic" manifestations,

4. Janet, P.: *Les obsessions et la psychasthénie* 2:7, 1911.

5. Barker, Lewellys: *The Clinical Diagnosis of Internal Diseases* 3:592, 1916.

6. Hitschmann, Edward: *Freud's Theories of the Neuroses*. Nervous and Mental Disease Monograph, Series No. 17, New York, Moffart Yard & Co., 1913.

opinions differ. Some, by virtue of tradition, or the therapeutic ease of treatment with a formula, apparently continue to think of these psychopathologic findings in terms of exhaustion and increased nerve irritability. Others who are bolder and more energetic offer ingenious psychologic explanations for these data. All draw a definite line between functions of body and functions of mind.

The cases on which this paper is based are not presented for the sake of rehabilitating one syndrome or welcoming the passing of another; nor are they intended to stimulate controversy by the introduction of further terminology. In reviewing the case data in the table and its elaboration in certain instances, it is inevitable that one reader will see only neurasthenics, and another only psychoneurotics, while still a third will notice only the wealth of psychoanalytic material that has gone to waste. The writer has had but one idea in mind, and that is to approach each case as a problem by itself; to describe faithfully the facts that it presents, and then to study them, not for the purpose of making them fit the Procrustes' bed of an etiologic theory, or set mode of treatment, but to study them for the purpose of ascertaining what these facts mean in the patient's life. In what setting of life experiences and constitutional makeup, as well as biologic activities, do they occur? What opportunities for modification do they offer, not only from the standpoint of the individual's metabolism and hygiene, but also from the standpoint of his constructive constitutional assets, his adaptive resources, his material for instinctive readjustments? With this aim in view it becomes useless to haggle over what symptoms should be charged up to mind and what symptoms charged up to body. The main concern of the physician is, or should be, the functioning of the individual as a whole. The expressions of maladjustment in this functioning are legion. Disorders of function cover a domain too wide to be divided by such artificial boundaries as mental and physical. They manifest themselves in facts of nutrition, mentation, internal secretions, metabolism, sex conflicts, growth, feelings of inferiority, circulation, bacterial and neoplastic invasion, emotional instability, toxic processes, poor adaptive qualities, etc. Each group of facts is capable of furnishing exactly the same reliable information to the scientifically trained observer, whether it may have to be studied through the medium of behavior and life experiences as an emotional state, or whether it may have to be studied through the medium of a chemical solution, or polariscope, as a metabolic fact. In any case, hunting down the disturbance of functioning in a human being is not so delightfully simple as the public, and alas! too often the physician, seems to think, even when the patient points an accusing finger of symptoms to some group of

viscera such as the gastro-intestinal tract, for example. Campbell,⁷ in his recent study of some thirty cases from the cardiovascular service of the U. S. Army Hospital at Lakewood, N. J., has shown what a complexity of factors such as "instinct, emotion and personality" are involved in many cases of disturbed heart functioning. Adolf Meyer⁸ was the first to emphasize "the missing chapter of ordinary physiology and pathology, the chapter dealing with functions of the total person, and not merely detachable parts." To this chapter he applies the term psychobiology, not in contrast to, but as "a special level of biological activity."

DESCRIPTION OF AUTHOR'S CASES

These sixty cases of invalidism presented for consideration are admissions to the Henry Phipps Psychiatric Clinic of the Johns Hopkins Hospital from 1913 to 1918. Fifteen of the number were untreated through lack of the patients' cooperation. Some refused to remain because they resented being on the psychiatric service, but the majority left when given to understand that their exclusive salvation did not lie in a continuance of drug and operative therapy. Of the 45 patients treated, 16 were discharged as "well," and have had no further return of their former symptoms; 25 were discharged as "improved," and 4 as "unimproved." It was not possible to trace 11 of the "improved," but of the remaining 14, 6 are completely well, 4 are well enough to work, although they still have a few complaints, and 4 have relapsed into their former invalid condition. As to the 4 discharged "unimproved," 2 are said to be well at the present time, 1 is at another hospital, and 1 it has not been possible to trace. Among the forty-five patients treated the average duration of symptoms is five years; the average length of their stay in the hospital is twelve weeks. The ages of the entire group of sixty patients vary from 20 to 72 years. The proportion of females to males in the series is about 3 to 1.

Symptoms.—Their complaints included headache, dizziness, general weakness, nausea, vomiting, abdominal pain, eructation, "acid" stomach, diarrhea, flatulence, epigastric distress, anorexia, constipation, exhaustion, general pains and burning sensations over the body, insomnia, profuse perspiration, quivering and shaking feelings, back-ache, "falling spells," numbness, pseudo-aphasic attacks, and smothering and choking sensations. These symptoms occurred singly or in various combinations and permutations of distress, resulting in all

7. Campbell, C. Macfie: The Rôle of Instinct, Emotion and Personality in Disorders of the Heart, *J. A. M. A.* **71**:1621 (Nov. 16) 1918.

8. Cases Nos. 7 and 18 were discussed before a joint meeting of the Washington Psychiatric Society and the Maryland Psychiatric Society, March 28, 1917.

degrees of incapacitation from the cheerful ambulatory invalid quite resigned never "to know a well day again," to the individual who had practically not been out of bed for nine years.

Examination.—Every patient was subjected to the same routine examinations on admission, which are as follows: A general physical examination including a complete neurological status, and an examination of the reproductive apparatus, together with laboratory studies of the urine, blood, etc. A blood Wassermann was made on every patient. These inquiries were supplemented by consultation with other clinics in the hospital such as the dental, roentgen ray, electrocardiographic, etc., and by such special investigations as gastric analyses, blood-sugar determinations, blood cultures, etc., according as the facts of the routine examinations or the patient's individual complaints indicated further research. The psychopathologic data were derived from a record of the individual's mental status, and especially from a study of the facts of his growth and development from childhood up with particular emphasis on the constitutional makeup and reaction tendencies. The patient was then given a frank report of the results of the various examinations, and was invited to think of his incapacitation, not as a disturbance in functioning of his gastro-intestinal tract, or some other point of visceral strategy from which his symptoms seemed falsely to emanate, but to think of it as a disturbance in the behavior and activities of his whole personality; he was urged to think of his symptoms as substitutes for reactions to unhappy experiences, thwarted ambitions, petty jealousies, romantic disappointments, an empty and dissatisfied life, a desire to escape marital or domestic responsibilities, or whatever the specific facts may be. While the patient was being encouraged to educate himself in this concept, he was given the benefit of any hygienic lifts suggested by the various examinations, such as attention to the weight curve, hemoglobin, eye-strain, etc.

METHOD OF TREATMENT

Equally essential as a reconstructive factor is the ward routine arranged so that the patient gets a full and well-ordered, but not an ostensibly planned day. Following breakfast in the dining-room there are two hours in the gymnasium and hydriatic department. The afternoon is broken into by the occupation class of an hour or more in weaving, basketry, carpentering, book-binding, etc., varied according to season and time by outdoor games, dancing and parties in the recreation hall, on the roof garden, or time spent on the campus. Between scheduled hours there is opportunity for the patient to sew, crochet, embroider, write letters, read current magazines, daily papers and library books, or to lie down if he chooses. These concrete activi-

ties not only form a sort of natural bridge between the self-limitations of the invalidism and the return to normal action and interests toward which the patient is headed, but they also restore confidence in his somatic capabilities in general, and especially in certain viscera against which he has so long nursed suspicions of incompetence. He becomes stealthily aware that he can eat proteins, carbohydrates and fats without disaster; that he can use his eyes without headaches; that he can exercise without fatigue and join a noisy frolic without collapse. And, with the knowledge that he can, comes usually the desire to do. The entire process of better adaptation is helped along by a conscientious sensible survey of the facts of the case with the physician, usually in personal interviews in the physician's office, reviewing the daily events, reactions and the observations by the nurses, giving consideration to the patient's complaint and problems, and removing them from the domain of vague self-indulgence to a reduction of concrete events and problems of the present, past or future. So much for the method of practical approach which has been employed with these patients.

In presenting the following data by table one can give only the roughest sketch of the actual facts of each case, and that sketch in a way that makes it hard to transcribe the human element in the individual situation. Each record covers the patient's age, sex, complaint and its duration, previous treatment, somatic condition, constitutional makeup, psychogenic material, condition on discharge and catamnesic data. A glance at the column of "psychogenic material" shows how varied are the "adaptations" for which the invalidism serves. The cases which follow describe a few of these mechanisms in detail.

REPORT OF CASES

CASE 1.—History.—No. 7 was an unmarried girl of 24 who was transferred to us from the medical service of the Johns Hopkins Hospital in June, 1916, with the diagnosis of neurasthenia. She was born in England of probably normal stock, and after a common school education took up the occupation of nursery governess. In 1913 she came to this country, and after living with her sister for a year took a nursery position in October, 1914, in a family in which she gave excellent satisfaction. While in the mountains with the family in the summer of 1915 she developed abdominal pain, nausea and occasional vomiting. She was treated for mucous colitis by a general practitioner, but without relief. In December, 1915, a surgeon removed her appendix. On recovering from the operation her symptoms returned and her employers took her to a specialist in internal medicine who treated her for hyperthyroidism without improvement. In May, 1916, she entered our own medical service where the physical and laboratory examinations were negative in every respect. She was transferred to the psychiatric service where the following psychogenic material was brought to light: The patient was a shy, sensitive child who got on so badly at home that she left at 18 to earn her own living. She came to this country in 1913 at the invitation of a married sister. On arrival she

found that this sister had left her husband and was living as the mistress of a married man by whom she was then pregnant. The patient was tremendously upset by the revelation. She brooded over the family disgrace but dared not write home about it. A sense of duty compelled her to remain with her sister until the latter's confinement. This period of mental distress was aggravated by her sister's bitter complaints of abdominal discomfort, nausea and frequent vomiting. During the patient's subsequent employment as nursemaid she was haunted by the fear that her employers would learn of her sister's disgrace, and also by the fear that her sister might again become pregnant. On the way to the mountains the following summer Pullman arrangements were such that the patient was asked to share her berth with her employer's little boy of 5 years who was a bed-wetter. She spent a tearful and sleepless night, and shortly after developed the gastro-intestinal symptoms described above, although there was no notion of an imaginary pregnancy on her own part, nor yet any conscious association of her own symptoms with those of her sister.

Course in the Hospital and Discussion.—With a thorough discussion of her relations with her sister of which she had never spoken to any one before, an elimination of the sister from the whole situation, with a reconstruction of the patient's ideas along her instinctive lines from an attitude of shame-faced mystery to a common sense dealing with concrete issues, the patient gradually lost her symptoms in six weeks. In spite of the daily routine of gymnasium, outdoor exercise, occupation class, etc., she gained 12 pounds. She was encouraged to carry out an ambition to take nurses' training which she had cherished for many years, and was accordingly discharged in August, 1916, to take up her new duties. This spring she graduates at the head of her class in the training school. The patient has never had any return of her old symptoms, or any other complaints in spite of the severe strain which she has undergone during the past three years.

CASE 2.—History.—No. 18 was an unmarried woman of 33 who was admitted to the clinic in July, 1916. Since 1904 she had been subject to diffuse headaches usually accompanied by nausea and vomiting, and frequently followed by "falling spells" without loss of consciousness or other signs suggesting an epileptic syndrome. At first headaches and "falling spells" came once or twice a year, but gradually increased in frequency and duration until at the time of admission they occurred once in two weeks—the headaches lasting four days. In 1911 she was obliged to give up her work as telephone operator, and from that time on had lived the life of a recluse because of her increasing invalidism. From 1913 to 1915 she was treated for epilepsy with bromids, but without improvement. On admission the physical examination was negative, including the neurological status, blood Wassermann test or any condition of malnutrition. There were no hysterical stigmata. The hemoglobin was 90 per cent. Shortly after admission the patient had two "falling spells" in which she gently slid to the floor, and in reaction to the mouth gag about to be used, said, "Don't do that, I'm all right."

Family History.—The patient's family history is negative. She was a bed wetter until 6 years old, and was always a timid and self-conscious child. She was brought up in a narrow, rigid home environment, where every normal instinct for recreation, social outlets and the expression of her individuality in any form was strictly repressed by a mother who dominated the household in every detail. The patient reacted to this atmosphere by a marked feeling of inferiority. She shrank from meeting people, seldom went out,

AUTHOR'S CASES

No., Sex, Age	Complaint	Duration	Previous Treatment	Somatic Condition	Constitutional Makeup	Psychologic Material	Stay in Hospital	Discharge	Comments
1 F. (W.) 72	Headaches	12 yrs.	Drugs (delirium)	Negative	Ego-centric, easily worried	None elicited	1 mo.	Unimproved	Not traced
2 F. (Sep.) 53	Headaches	13 yrs.	Best cure; pelvic operation; operation on muscles of accommodation; calomel therapy to stomatitis	Stomatitis, anacidity, under-nourished through self-starvation	Sensitive, anxious, nagging disposition	Alcoholic husband described her 3 years after marriage, the son is epileptic	3 wks.	Improved	Well for 4 months, then a gradual relapse
3 M. (S.) 95	Headaches	1 yr.	Best cure; 3 nasal operations and a tonsillectomy	Negative	Jealous, grouchy, easily slighted	Jealous of brothers, resentful for her step-mother for her supposed partiality; constantly at friction with employer	3 wks.	Untreated	Patient is over-keen with engineering work; no other ailments
4 F. (S.) 25	Nausea, insomnia, general weakness; pains over body	2½ yrs.	Three rest cures	Negative	Insincere, whining, un-energetic	Intolerable home situation; father denied her ambitions for further educational or social outlets	7 wks.	Improved	Patient took up a business course; still has a few complaints
5 M. (M.) 52	General pains	2 yrs.	Several rest cures	Pyorrhea, (chr. proctitis, moderate hyper-tension	Shy, untrusting, worthy, ego-centric	Jealous of his wife; worry over sex irregularities, and at least one illegitimate child	1 wk.	Untreated	Not traced
6 F. (Sep.) 30	Headaches	16 mos.	None	Chronic tonsillitis; retro-position of uterus	Superficial bravado attitude; insincere	Separated from husband because she was jealous of her mother-in-law; great deal of sex tension; openly declared she used to escape thinking of the past	1 wk.	Untreated	Not traced
7 F. (S.) 24	Nausea, abdominal pain; vomiting	1 yr.	Treated for mucous colitis; thyroid disease; appendectomy	Negative	Shy, timid, "nervous" child; efficient worker	Worry over sister's illegitimate pregnancy; nursed sister who complained of nausea and abdominal pain	3 mos.	Well	No return of symptoms; patient has finished nursing course
8 M. (M.) 49	Headaches, weakness; stomach trouble	3 yrs.	Nasal operation	Negative	Impulsive, no self-control	Great sexual promiscuity; deserted his wife; symptoms referable to sex conflicts	4 wks.	Improved	Not traced

W., widow; Sep., separated; M., single; M., married.

9 F. (M.) 43	Stomach trouble; pain and burning sensation	18 yrs.	Gynecologic operation; 9 rest cures	Röntgen ray shows dilated ach; total acidity, 45	Excitable, restless, easily worried	Onset of symptoms after birth of second child; patient is nagging and jealous of husband; has no occupation, ambition, or interests outside herself	3 wks.	Untreated	Not traced
10 M. (M.) 32	Insomnia, flatulence, indigestion, pain in heart	6 mos.	Family physician	Negative	Emotionally unstable	As a child patient was dominated by mother; his wife is shallow, takes no interest in home, neglects children, is unfaithful	7 wks.	Well	Continued well; has worked every day since discharge; no complaints
11 F. (M.) 46	Erectation pain and burning over body	24 yrs.	For hysterectomy; constant medical attendance	Infiltration of both apices	Domineering, evasive, self-centered	No interests outside her complaints; husband does not give her enough sympathy and attention	2 wks.	Untreated	Her doctor writes: "Patient looks well but begins to complain when she sees me." Back at work, but still has a few complaints
12 M. (W.) 43	Weakness of legs	1 yr.	Continuous genito-urinary treatment for 1 year	Chronic urethritis	Stubborn, rigid, full of self-pity	Neisser infections, illicit intercourse with moral conflicts since death of wife seven years previous; patient made locomotion the scapegoat of his conscience	3 yrs.	Improved	
13 M. (M.) 47	Insomnia, pain in head and back	10 yrs.	Diets and medicine all the time	Slight protheca varicocele in left scrotum; total acidity, 20	Seclusive; easily worried about himself	Patient's symptoms were coincident with foreclosure of mortgage on his farm	2 wks.	Untreated	Not traced
14 F. (W.) 54	Palpitation, photophobia, throbbing over body	9 yrs.	Rest cures, bed-ridden 6 years	Low grade nephritis	Lazy, self-indulgent, hypocritical	Noise ever elicited; symptoms appeared suddenly after husband's return from a vacation she had opposed	3 mos.	Well	Patient does all her housework; no complaints at all
15 F. (W.) 32	Headaches, insomnia, anorexia	2 yrs.	Rest cures, drugless healer	Negative	Studious, imaginative, inaccessible	Three difficult labors; patient dreads more children; invalidism a protective role	5 days	Untreated	Not traced
16 M. (M.) 45	Weakness, headache, dizziness	4 yrs.	Dispensary habit; four rectal operations	Chronic nasopharyngitis	Unstable emotionally	Illiterate immigrant, 21 years in U.S.; wife, 3 sickly children; constant financial struggle	5 days	Untreated	Works every day but is depressed and tearful
17 F. (M.) 32	Insomnia	3 yrs.	At least four different doctors	Underweight; Hb, 70 per cent.; achylia gastrica	Neurotic child, whining, dependent	Marrried against partner's wishes; continuous feud with her mother; patient disinherited; strong father attachment with depreciating comparison to husband	7 wks.	Improved, gained 12 pounds, Hb. 90 per cent.	Not traced

AUTHOR'S CASES—Continued

No. Sex. Age	Complaint	Duration	Previous Treatment	Somatic Condition	Constitutional Makeup	Psychogenic Material	Stay in Hospital	Discharge	Catastrophe Data
18 F. (S.) 33	Headaches, nausea, "fainting spells"	12 yrs.	Bromide treatment for 3 years	Negative	No self confidence, dependent; puritanical sense of duty	Inferiority complex aggravated by narrow home environment and a hard-headed dictatorial mother; no divisions	3 mos.	Well	Patient has worked every day since discharge; no complaints
19 F. (W.) 32	General acidity	6 yrs.	Diets to starvation; autointoxication therapy; tuberculosis specialists	Fibroma of larynx; undernourished; visceroptosis	Self-confident, positive	Symptoms were coincident with death of her husband; life since then has been narrow and shut-in; patient has devoted herself to nursing invalid mother and valid mother and winning sisters	1 mo.	Improved	Practically well
20 M. (M.) 41	Weakness, regurgitation of food	2 yrs.	"Doctoring" for indigestion	Splanchnoptosis	Shy, sensitive, easily embarrassed, accusative	Gradual decline of efficiency, could not hold jobs, sexual difficulty	3 mos.	Improved, gained 21 pounds, Hb. 80% improved	State hospital with depression for 4 months; not traced further
21 F. (S.) 56	Abdominal pain so can't walk, gas, vomiting	4 yrs.	Appendicectomy; appendectomy and resection of colon, rest cures between operations	Emaciated from self-starvation, Hb. 70 per cent.; Bell's palsy or right rectovaginal fistula	Rigid, dominating, self-righteous	No cooperation with analysis; patient's expenses are paid luxuriously by a sympathetic wealthy friend	5 wks.	Improved	Two operations since discharge, now a rest cure
22 F. (S.) 35	Restlessness, palpitation	3 yrs.	Two operations for thyroid trouble	Negative	Neclusive, reserved	Patient in love with a married man	1 mo.	Improved	Stayed at Sheppard and Pratt Hospital, now at home of sister who says she is well
23 M. (S.) 28	Weakness, stomach trouble, exhaustion	4 yrs.	Continuous rest cures, gastric lavage, diets; genital-urinary douches	Dental caries; some fibroid changes in lungs	Shy, poor mixer, sensitive, ascetic	Sex conflicts, masturbation with inferiority feeling since 14 years; obsessive tuberculosis fears; two broken engagements	3 wks.	Improved, gained 10 pounds	Well, working every day
24 F. (M.) 40	Insomnia, anorexia	5 mos.	Gynecology operation (D. and C.); rest cure	Undernourished, pyorrhea	Limited original equipment, easy worrier	Symptoms coincident with death of mother-in-law and a miscarriage; life of farm drudgery; husband a hypochondriacal depression	5 wks.	Improved, gained 6 pounds	Not traced
25 F. (S.) 37	Backache, insomnia, quivering attacks of pseudophasia	14 yrs.	None	Chronic tonsillitis; blind for 19 years	Ambitious, shy, sensitive	Secret rebellion against her blindness; constant worry; over father's alcoholism and terrible fear of forgetting the power to visualize; resented blindness as thwarting her ambition	3 mos.	Well, gained 15 pounds	Well, complete relief from previous symptoms

26 M. (M.) 27	Dizziness	1 yr.	Different doctors	Negative	Overconscientious	Symptoms coincident with discovery of wife's pregnancy; work fretted him; lost confidence in himself	3 wks.	Well	Well, no further complaints
27 F. (S.) 26	Backache	4 yrs.	Special corset; treatment for floating kidney	Secondary anemia	Empty, evasive, uncooperative	Conflict over sex tension, erotic episode with smooching of the incident	2 wks.	Unimproved	Well
28 M. (M.) 44	Dizziness, weakness, headache	1 yr.	None.	Negative	Alcoholic, floating labor class	Married twice; first wife unfaithful; no interest outside of his complaints; craves sympathy	9 wks.	Well	Practically well, works every day but is morose and whining
29 F. (M.) 37	Headaches, exhaustion, gas	14 yrs.	Rest cure	Negative	Clinging, whining, self-pity, "poor stuff"	Afraid to have children; weakness coincident with miscarriage in 1900; head aches since; sister died of brain tumor in 1909; spoiled by husband	5 wks.	Improved	Well, does all her housework
30 F. (M.) 31	Headaches	5 yrs.	Three rest cures, 2 nose and throat operations, 1 dental operation; oculists rest cures, nasal operation	Negative except for otosclerosis	Dependent, timid, craving sympathy	Worry over fear of inheriting insanity; broken engagement; jealous tendencies; dread of childbirth	9 wks.	Well	Well
31 M. (M.) 38	Stomach trouble	3 yrs.	Diets, rest cures, nasal operation	Hypertension, acidity due to limited diet	No sense of humor, sober-minded, constant worker	None elicited, talking constantly of symptoms; dissatisfied	10 wks.	Improved	Not traced
32 F. (S.) 30	Constipation, flatulency	2 yrs.	Diets, rest cure, osteopathy	Negative	Self-exacting; hypochondriacal	Complaints and substitutes for thwarted ambitions; repressed sex longings with prudishness and religious compensation	4 mos.	Well	Well, has completed library course and is working
33 F. (M.) 38	Pains in thighs and jaws and "brain"	16 yrs.	Six gynecology operations and treatments, osteopathy, electricity, teeth out	Negative	Shallow, nagging, stubborn	Symptoms followed birth of her first and only child	6 days	Untreated	Not traced
34 M. (M.) 39	Weakness, headache, insomnia	1 yr.	Family doctor	Negative	Shy, poor mixer; dissatisfied	Ambitious beyond assets; day dreams; writing plays instead of job he can do; symptoms began one month after marriage	4 mos.	Well	Well
35 F. (M.) 33	Belching, exhaustion, weakness, flatulence	5 yrs.	Two rest cures, appendectomy	Slight hypochlorhydria	Unstable, capricious, dependent	Wants no more children; strong sex desire; husband cold and unsatisfactory; no interests outside self	3 wks.	Well	Well

AUTHOR'S CASES—Continued

No. Sex Age	Complaint	Duration	Previous Treatment	Somatic Condition	Constitutional Makeup	Psychogenic Material	Stay in Hospital	Discharge	Outstanding Data
36 F. (M.) 24	Ab-normal pain back-ache	3 mos.	Eight tube and ovary removed	Negative	Shallow, sentimental, selfish	Complaints used to avoid further pregnancies; they began after 3d abortion	3 wks.	Improved	Not traced
37 F. (M.) 42	Headaches	9 yrs.	Morphia; two gynecologic operations drainage of gall-bladder	Negative	Rigid, prudish, self-acting	Headaches are due over husband, no desire for children; dis-	6 wks.	Well	Well but resentful of analgesic
38 F. (S.) 60	General aches and pains, twitching over body	12 yrs.	Two gynecologic operations, many doctors	Ventral post-operative hernia; dental caries	Morbid, narrow, easy worrier	Appointed in life; has spent life with no interests but teaching and waiting on invalid mother and sister	5 mos.	Improved	then about house work, but in full of complaints
39 M. (S.) 24	Dizziness, constipation, weakness	1 yr.	Family doctor	Negative except pyorrhea	Suggestible, timid; no initiative	Neurotic home environment; father has gastric neurosis; probable sex conflicts	1 mo.	Improved	Not able to work
40 F. (S.) 62	Backache, dizziness	40 yrs.	Dieting and rest	Hypertension; chronic fibroid changes in lungs	Sensitive, dependent, timid	Symptoms coincident with breaking of engagement at 31 years; invalid since; symptoms worse since attack of acute Bright's disease at 40; no intervals	1 mo.	Unimproved	Mansturlum
41 F. (M.) 37	Headache and acid stomach	25 yrs.	One gynecologic operation, five rest cures	Hydrochloric acid deficit of 10	Diffident, craves sympathy	Mother, father and sister all had headaches; patient jealous of sister; husband is unsympathetic; she does not want children	1 wk.	Untreated	Not traced
42 M. (S.) 34	General "poor health"; weakness	All life	Was taking nine different medicines on admission	Negative	Morbidly self-centered, full of self-pity	Patient was brought up to believe he was defective; was taken out of school, was not allowed to work	1 wk.	Untreated	Not traced
43 M. (S.) 26	Weakness on exertion, pains all over	8 yrs.	Diapneusies	Left inguinal hernia	Erratic, never could hold a job	Patient had two schizophrenic breaks at 18 and 22 years, probably using his complaints to keep him from worry; masturbation; worry	3 mos.	Improved	Not traced
44 F. (S.) 48	Headaches, vomiting	1 yr.	Rest, cure, daily gastric lavage for 1 year	Negative	Inclined to self-pity, craves sympathy	Symptoms coincident with hard nursing case; life of drudgery to support mother and sister; no recreation or emotional outlet	6 wks.	Well	Well

45 M. (M.) 52	Weakness, exhaustion	6 yrs.	Rest in bed	Negative (hay fever)	Sensitive, easily discouraged	Heavy financial burdens; marital incompatibilities	4 mos.	Untreated	Not traced
46 F. (S.) 41	General aches and pains; cannot use eyes	8 yrs.	Two rest cures; 1 gynecologic operation, electricity	Refraction error; anacidty	Sensitive, immature, stubborn	Symptoms coincident with father's death in 1910; autoeroticism with worry	3 days	Untreated	Not traced
47 F. (S.) 26	Pressure and pain in head	5 yrs.	None	Undernourished; poor hearing in left ear	Somatically interested, dependent, craving sympathy	Symptoms associated with fear of high blood pressure; they developed at time of brother's engagement as partial adaptation to elicit sympathy and prevent his marriage	6 wks.	Well	Well
48 M. (S.) 38	Weakness	7 yrs.	Two rest cures; 1 nasal and throat operation; operation for gastric ulcer (not found); dispensaries	Negative	Self-centered	Parent's mother, brother and sister are invalids of neurotic type; does not want insight into illness; his disability insurance runs out; he insists on rest	1 mo.	Untreated	Took a rest till insurance stopped; is now working but full of complaints
49 F. (M.) 40	Sweating, burning, aching	5 yrs.	Two operations, (perineal repair, hemorrhoids, appendectomy) osteopathy	Negative	Stubborn, lazy, self-indulgent	Financial reverse; no desire for children; symptoms appeared after last pregnancy	4 mos.	Improved	Not traced
50 M. (M.) 39	Headaches	2 mos.	Dispensaries and outside doctors	Pyorrhea; impairment of hearing	Neurotic; Russian Jew type	Symptoms coincident with abscess in throat, and parents leaving city; father has headaches; financial straits	5 wks.	Improved	Well, working steadily
51 M. (M.) 33	Dizziness, weakness in limbs	1 yr.	Medicine and rest	Accidental loss of eye in childhood	Sensitive, introspective, easily hospitalized	Constant fear of disease; symptoms coincident with mother's death	8 mos.	Improved	Not traced
52 M. (S.) 33	Belching, indigestion	12 yrs.	Various doctors	Negative	Neurotic child, backward, sensitive	Marked inferiority feelings with autoeroticism; father is a gastric invalid	6 days	Untreated	Not traced
53 F. (M.) 29	Pain around heart; stomach trouble	7 yrs.	Gastric lavage every 10 days for 3 years; two gynecologic operations	Negative	Precise, hypochondriacal	Symptoms coincident with jealousy of husband; complaints called for his sympathy and attention	5 mos.	Improved	Complaining but able to get about her home duties
54 F. (M.) 35	Stomach trouble, weakness	2 yrs.	One gynecologic operation	Negative	Dependent, weakling	Eight periods of invalidism of from 2 to 8 months since 17 years	7 wks.	Improved	Not traced
55 F. (M.) 39	Shaking and quivering	10 yrs.	Medicines and doctors	Negative	Innocent, evasive	Worry over daughter's mental breakdown	2 wks.	Improved	Well, no return of symptoms

AUTHOR'S CASES—Continued

No., Sex, Age	Complaint	Duration	Previous Treatment	Somatic Condition	Constitutional Makeup	Psychogenic Material	Stay in Hospital	Discharge	Catamnestic Data
76 F. (S.) 20	Nausea, belching, epigastric pain	3 yrs.	Treated for stomach trouble	Negative	Lazy, stubborn, over-indulged	Symptoms coincident with having her teeth out, would not have new ones, sensitive	6 wks.	Improved	Works every day but still enjoys a few complaints
57 F. (M.) 30	Pain in side	2 yrs.	One gynecologic operation, treated by six doctors for gastric ulcer and Diet's crisis	Negative	Jealous, subject to temper tantrums, vain, shallow	Anxiety, neurosis at 15 years, pain spasms precipitated by tantrums with husband; latter has been promiscuous since marriage	5 wks.	Well	Well
58 F. (S.) 25	Smothering feeling	5 yrs.	Various doctors	Secondary anemia, vitreoretinosis, detected nasal septum	Lazy, full of self-pity, rigid, pretentious	Symptoms coincident with broken engagement; disappointed educational ambitions; parasitic life for last 3 years; no desire for insight	3 wks.	Untreated	Not traced
59 F. (S.) 35	Headaches, weakness, pains in eyes	10 yrs.	Various doctors	Hemoglobin 60 per cent., undernourished	Shy, always complaining, easy worrier	Autoeroticism for 10 years; feared insanity from habit; invalidism a compensation for an idle, unsatisfactory life	2 mos.	Well, gain in weight and Hb.	Well
60 F. (S.) 30	Pain in back, side and stomach	2 yrs.	One operation (appendectomy)	Chronic otitis media	Self-indulged, clinging, craves sympathy	Patient curled up in presence of earning her own living, and has found it easier to live on sympathetic friends	2 wks.	Untreated	Not traced

suppressed all interest in the opposite sex in conformity to her mother's wishes, and feared to take responsibility so that she refused several chances to advance while working as telephone operator. The patient's first "falling spell" occurred in 1904 when at the time of the Baltimore fire she came unexpectedly on the charred body of a neighbor. For the next three or four years these attacks and headaches always followed some exceptional physical or mental strain, but from 1910 they occurred irrespective of any unusual circumstances, seeming to represent the patient's only means of getting square with an intolerable home situation.

Course in the Hospital and Discussion.—In going over matters with her these facts were clearly pointed out. She was told that there was no organic basis for her complaints, that they were expressions of her inner conflicts and dissatisfactions, and that the solicitude and sympathy called forth by her affliction probably satisfied in a large measure her natural craving for affection and the expression of her individuality. She was shown the necessity of gradually reconstructing her life along the lines of reaching out toward new outlets in the cultivation of friends, social interests, recreation, and above all some occupation which would give her satisfaction. The patient was discharged in October, 1916. She has never had any more "falling spells." The headaches have decreased, so that she has had but seven or eight attacks a year, and only one of these has been severe enough to keep her from an eight-hour day at the office where she has been employed since leaving us. She has been boarding in a congenial place in town, and has taken up various social activities. On her own initiative she has been attending regular classes at night school during the past winter with the view of improving her position.

CASE 3.—History.—No. 30 was a married woman of 31 who was referred to us in October, 1917, by Dr. Louis Hamman of Baltimore, whom she consulted because of headaches of five years' duration. The patient's mother is an erratic, nagging woman subject to temper tantrums, but aside from this the family history is negative. At 4 years of age the patient had a "fever" followed by a deafness which has grown progressively worse. At 19 she graduated from a normal school, after which she lived at home, substituting in local schools, but unable to get a permanent position on account of the deafness. In June, 1917, she married, but has not been pregnant. The patient's headaches began in 1912 shortly after hearing that her soldier brother was shot in the Philippines. She was sleepless, and lost her appetite, but put on a "bold front" plunging into athletics (tennis and golf) until she collapsed with "exhaustion" several months later. The attending physician put her to bed for nine months with absolute rest and forced feeding, hinting darkly at tuberculosis. During this rest cure her father died, and a brother returned from the West to die at home of tuberculosis. In August, 1914, becoming nauseated from milk and eggs (as she said), the patient got out of bed and consulted another physician in a distant city. He told her she had "thyroid disease." The patient returned home, continued to have headaches on the slightest exertion, and worried constantly about herself. From May, 1915, to November, 1915, she sought health in the mountains of North Carolina, but found a nose and throat specialist who promised instant relief from a drainage of the right antrum. This operation, however, caused no improvement. In April, 1916, she had a tonsillectomy, also without relief. In July, a dentist treated her for "pus pockets" and an impacted wisdom tooth. This, too, was a failure so far as relief from the headaches was concerned. The patient then began to consult oculists, four of whom changed her glasses. At that time,

she consulted Dr. Hamman and was recommended to the clinic. She could not read, sew or take exercise for fear of inducing a headache, which appeared on an average of once a week and lasted for twenty-four hours.

Physical Examination.—This was practically negative except for otosclerosis. There was a slight retroflexion of the uterus. Her glasses corrected a moderate astigmatism, myopia and some weakness of the internal recti muscles.

Constitutional Makeup.—The patient had always been self-centered, inwardly dependent, craving sympathy and attention, and full of self-pity, but outwardly altruistic and bluffing. The deafness has tended to increase her sensitiveness and to limit her activities and ambitions.

Psychogenic Material.—The following psychogenic material was brought to light during a study of the case:

1. The patient had never been able to face the fact that her soldier brother shot himself in the Philippines instead of being killed by natives as the family advertised. She felt that he had inherited insanity from her mother, who has violent temper tantrums. And if he had inherited it why should not she? Her headaches were interpreted vaguely as a possible forerunner of mental disease. A friend dying of brain tumor had complained of headaches.

2. The patient had always been overshadowed by a handsome, sparkling sister of whom she was secretly jealous. This sister received more attention in society than the patient. The latter felt not only slighted, but had marked feelings of inferiority based on her deafness. Even after marriage when she and her husband were boarding the patient was sensitive to her husband's attempts to make himself agreeable to others in the household.

3. During the beginning of invalidism the patient had been undecided whether she should marry a wealthy man to whom she was engaged, or her present husband who was struggling to rise in his profession. She did not love the first man but felt he "understood" her better (i. e., gave her more sympathy and attention), and would be better able to provide for her comfort.

4. The patient hesitated also about the whole question of marriage because she did not want children. She said it was because she feared they would inherit her "tendencies to insanity." In reality she feared childbirth because a sister had died of Bright's disease during pregnancy. Since marriage every doctor had told her she was not strong enough to have a child—at least, until her headaches were better.

Course in the Hospital and Discussion.—It is scarcely to be wondered at that the patient had been willing to remain passive toward her decisions and problems and to accept medical excuses that involved no effort on her part toward remedying matters; but it is a matter for marvel that after five years of invalidism under careful therapeutic supervision she had moral force enough to turn squarely about and face her difficulties frankly. Four weeks from admission she was reading books and magazines, embroidering, exercising in the gymnasium and taking three lip-reading lessons a week which were advised as a lift to make her more at ease in meeting people without embarrassment from deafness. The patient was discharged ten weeks from admission comparatively free from headaches, and eager to take a fresh start. She reports to the physician from time to time. Since leaving the clinic she has been doing all her own housework, much of her sewing, and has been going out normally with pleasure and satisfaction. Of her former complaints she says: "Yes, I have headaches sometimes, but they don't bother me any more. I can always find the reason why they came on that day."

One often sees invalidism growing like a fairy weed out of some stray seed of somatic disorder such as hypochlorhydria, retroflexion of the uterus, visceroptosis, a mild pulmonary tuberculosis, etc. With the disturbance corrected one would suppose the patient would give up his complaints. Not at all, he harps on them more than ever. The physician thinking he must have overlooked something, and goaded by the feeling that he ought to be "doing something" about all this distress, sends the patient from one specialist to another. It never occurs to physician or family to ask Of what is the patient making his complaints the scapegoat? Is it conscience, or instinct, or a natural tendency towards inertia? Why is it that this patient with a mild degree of visceroptosis is collapsed in bed with a special nurse, when another individual whose roentgenogram shows a stomach almost to the pelvic brim accomplishes a good day's work with a maximum of enjoyment and a minimum of discomfort? Obviously, we are dealing with a situation too complex to be explained by the visceroptosis. The following case is illustrative of this type of invalidism:

CASE 4.—History.—No. 23. A single man of 26 was admitted to the clinic in May, 1918, complaining of exhaustion, weakness, stomach trouble and fear of tuberculosis. The family history is negative. As a child the patient was timid and retiring, and shrank from every contact that might embarrass him. He finished high school at 16, graduated from college at 20, and began professional school which he left at 22 because of the present illness. Following an attack of bronchitis in April, 1914, one doctor told him he had tuberculosis and should give up his work; while others told him he was well and should continue it. The patient compromised by going West on a ranch to rest. He was, as he had been all along, afebrile and symptomless. In May, 1915, he had diphtheria, following which he had an afternoon rise in temperature to 99 F. for several months. In August, 1915, a doctor told him he had r les in both bases. In November, he went to a sanatorium where he stayed until February, 1916. The doctors here could find nothing wrong with him, but kept him in bed most of the time. In December, 1915, he complained bitterly of "heaviness" and acid eructations after eating; June, 1916, found him at another sanatorium where his physical examination was found to be negative. A gastro-intestinal specialist, whom he consulted in December, 1916, found hyperacidity, and put him on a "mild diet" and daily gastric lavage which the patient continued until June, 1917, when another specialist advised him to discontinue the performance. He did so and gained 20 pounds. In February, 1918, he consulted a genito-urinary specialist for nocturnal emissions, and began a course of treatment consisting of instrumentation and injections of protargol.

Physical Examination.—Physical examination at the clinic after admission showed dental caries, fibroid changes in the lungs but no active process, a slight hydrochloric acid deficit, and a hemoglobin of 73 per cent.

Psychogenic Material.—This consisted for the most part of a life-long sex conflict. At 14 the patient began the habit of masturbation, accompanied by feelings of sin and degeneracy. He brooded over the habit, felt that it was ruining his health, and thought that people could read it in his face. Through school and college he was a poor mixer, refusing invitations to join fraternities

and clubs, and restraining himself from making friends. He did not go in for athletics because he felt that the masturbation made him physically inferior to other men. From 1910 he was engaged to a girl who finally jilted him in the summer of 1917. The engagement period was one of a great deal of frustraneous excitement with numerous sexual episodes, following which there were exacerbations of autoerotism with tremendous worry and religious conflict. Each emotional and instinctive upheaval was associated with showers of somatic complaints. For example, the onset of the gastric symptoms in 1915 was coincident with the patient's fiancée throwing him over the first time.

Course in the Hospital and Discussion.—During the patient's three weeks in the hospital he gained 10 pounds. He was given the benefit of attention to the teeth, hypochlorhydria and hemoglobin. He was encouraged and reassured as to his physical condition and urged to prove his strength by walking and other exercise. At the same time the patient was shown how his fears of tuberculosis and his gastric symptoms were merely expressions of his instinctive conflicts. Into these facts he showed remarkable insight and availed himself of opportunities for frank discussion of his difficulties and of further suggestions for self-development. Since leaving the hospital he has been teaching physical training, and expects to resume his professional studies another fall.

CASE 5.—No. 14 is an example of the invalid from whom the physician cannot extract any psychogenic material although he is absolutely certain of its existence. These patients are usually persons who have indulged in life-long habits of mental dishonesty. Their life is "an open book" in which there is not a page of the past or present that they would not gladly have read. This insincerity of attitude is not infrequently glossed over with a superficial enthusiasm and interest in religion, philanthropy and the humanities, resulting in a surface so smooth and shining as to be disastrous to any honest attempt to get at the facts beneath. Curiously enough these patients often make good recoveries as in the following case, whose return to normal activities was due almost wholly to the training of the ward routine under stern and rigorous supervision. The case is significant not only because its outcome was not dependent on analytic lifts, but also because the invalidism itself was associated with a mild somatic disorder more or less chronic.

History.—The patient was a widow of 54, who was admitted to the clinic in June, 1917, complaining of headache, numbness, throbbing all over the body, epigastric pain after eating, and extreme sensitiveness to light and sound. Little was ever elicited in the way of family history or past history. The patient taught school for ten years previous to her marriage at 30. Friends describe her as "easy going" and inclined to self-indulgence and evasiveness. Her four children are bright and healthy. The present illness began nine years previous to admission. On the return of her husband from several months' vacation (the patient had opposed this trip), the patient took to her bed with all the above-mentioned complaints, though she seemed to be in good health up to that time. Her husband, who was a physician, sent her to a sanatorium for several months' rest cure. On her return she continued it until she was bedridden for six years before admission, causing herself to be isolated in quiet country places. During her invalidism she took no interest in reading, current events, domestic affairs or handiwork. She lay quietly in bed in a room darkened by closed blinds and drawn shades.

Physical Examination.—This revealed carious teeth and some edema of the feet and legs. The urine had a trace of albumin with a few hyaline casts.

The phenolsulphonephthalein test for renal function was 52 per cent. in two hours. Blood pressure was 135-85.

Psychogenic Material.—No information was ever obtained from the patient as to personal difficulties, or the setting in which her symptoms began. She always spoke of her husband with affection. He was a remarkably perfect man. Her children were ideal, and she the most devoted of mothers. When one applied to this devotion the touchstone of the fact that she was quite willing to deprive them of educational advantages and use the money to foster her invalidism, the patient glided easily into religious platitudes. Her sickness was "God's will." She had no worries or regrets "because I am a Christian."

Course in the Hospital and Discussion.—The patient refused dental attention. She was put on a low protein diet following which the edema disappeared and the blood pressure fell to 125-76. Although the patient never gave up her attitude of martyred suffering and her religious defenses, it was possible to pry her apart from her invalid habits one by one. The shades at the windows were raised, her blue glasses and the cloth over her eyes were removed. The cotton was taken from her ears. She was made to get out of bed, to walk first about the ward, and later over the building. She was required to take up handiwork, and be responsible for the reading of daily current events in the papers. At first constant vigilance on the part of the physicians and nurses was necessary to keep the patient from dodging these concrete occupations. No opportunity was lost to make it clear to her that she was using her invalidism as a means of escaping the responsibilities of life. While she did not respond to such proddings with denial or even resentment, it was noticed that she gradually showed more spontaneity in getting into the swing of activities. On discharge three months after admission, the patient was enjoying a full ward day. Since returning home she has taken up the management of the household, doing most of the housework herself.

Another type of invalid not only discouraging from the standpoint of presenting a "dry tap" so far as psychogenic material is concerned, but also discouraging from the standpoint of modifiability, is a patient like No. 38. These persons manage to adjust themselves somehow or other to the obstacles that confront them on the journey of life until they get to a certain point which very frequently is middle life. This adjustment has always been more or less superficial. In fact the patients seem constitutionally self-centered, having no healthy, robust interest in, or knowledge of, the world around them except indirectly through the medium of their family or own narrow circle of friends. A petty ailment in themselves or their families, a disappointment, or even a slight domestic inconvenience plunges them into a worry and fussing out of all proportion to the discordant element. As they approach the fifties, and the area of their interests and satisfactions becomes progressively more circumscribed—perhaps by the death of husband or wife, or the marriage and leaving home of children—they fall into a monotonous harping on vague somatic complaints for which they travel from one doctor to another. As therapeutic problems for

modification they are difficult, indeed. Robbed of their complaints the emptiness of life leaves little to which one can bring them back, and life-long poverty of interests makes it almost impossible to open up new avenues of satisfaction as substitutes for the old unhealthy reactions. The hospital regimen improves them a bit, but no sooner are they out than a slump is well-nigh inevitable.

CASE 6.—History.—The patient, a single woman of 60, was admitted to the clinic in May, 1917, complaining of jumping and twitching sensations in her arms and legs, pains in her eyes, and a "hollow feeling in her liver" that forced her to take great sighing breaths. As a child she had night terrors. At 18 she graduated from high school and had taught in public schools for twenty-five years until retired on a pension in 1909. At 18 and 20 years of age she had attacks of "neuritis" in her head and neck. In 1906 she had uterine fibroids removed, and in 1909 she suffered a panhysterectomy. The patient was always sensitive and inclined to worry over physical ailments for which she was continually consulting doctors. Since 1916 she had been complaining as on her admission to the hospital.

Physical Examination.—This revealed a postoperative ventral hernia, injected tonsils and decayed teeth.

Psychogenic Background.—The patient took no interest in any attempt to analyze the situation in which her invalidism occurred. She had spent all her life teaching in the grades, and caring for an invalid mother and sister. Since her retirement from teaching she had lived in an out-of-the-way country spot with her sister. Her life had been devoid of interests and satisfactions outside her own home circle. She had cultivated no outlets in the way of reading, recreation or social intercourse. Everything in her manner of living had tended to foster her innate tendency to withdraw from the outside world into herself.

Course in the Hospital and Discussion.—The patient refused dental attention. The gynecologic consultant thought the ventral hernia quite insufficient to account for her complaints, and recommended an abdominal support. During her five months in the clinic she gained 20 pounds, and slept without difficulty. She showed oscillations in her complaints according to how vigorously she was prodded into activities. Any mention of a time limit to her stay in the hospital was sufficient to precipitate them in showers—she became tearful, and would declare she had come "to be cured and not helped," and thought her case ought to be tried out for eight months. At other times the patient would show an easy, placid hospitalization. There was never any acceptance of the obvious adaptive rôle of her invalidism. Since leaving the clinic she has been living at home. She makes her usual complaints but apparently is able to adapt herself to some household duties.

The above records represent merely a few of the psychobiologic twists for which invalidism may be a substitute. Each case in itself is a problem for study. The physician's task is not at an end when he has brought to light the psychopathologic facts underlying these cover reactions. Why is it that ordinary "slings and arrows of outrageous fortune" cause a break in the adaptive compensation of these particular individuals? And why should this break in compensation

take the form of an invalid reaction rather than one of the major psychoses? These and kindred questions involving a working knowledge of the problems of psychobiology should be a part of the medical equipment of every practicing physician as much as *materia medica*. A glance at the medical profession's groping efforts after salvation recorded in the "previous treatment" column of the appended table is enough to convince the most prejudiced of this necessity. As servants and medical advisers of the public it is our duty to study carefully *all* the facts of individual disease brought to our attention, and to base an opinion on these facts unbiased by the habit training of a specialty or the conservatism of tradition.

STATUS LYMPHATICUS: ITS OCCURRENCE AND SIGNIFICANCE IN WAR NEUROSES

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In the study of the psychoses, perhaps most particularly in the study of dementia praecox, the investigation of the makeup of the individual has meant great progress both as regards broad lines of diagnosis and sympathetic understanding of individual cases. These studies of makeup can be said to be preponderately along psychic lines. They are actually personality studies, and as such sometimes include a general survey of physical factors. However, it is fair to say that usually the psychic tendencies are studied without even a recording of the physical traits to be correlated with them. Considerations of this sort regarding a recognized advance in the study of a psychotic type, contemporaneous with meeting for the first time in fairly large numbers cases of war neurosis, led the writer to study makeup in connection with the occurrence of the war neurosis. However, the study of the personality in these cases was not undertaken, the psychic makeup was not established and no conclusions drawn from that realm. The investigation was frankly restricted to the physical. Perhaps the fact that the war neurosis is new only as regards its physical stage setting made it natural to look at the physical attributes of its victims. The physical attributes searched for have been those that occur in the condition which is called status lymphaticus. These physical signs will be enumerated and their bearing on the war neurosis discussed. Unfortunately, the concept status lymphaticus does not in every sense bear scrutiny and the writer is anxious to disclaim having found cases that displayed all of the signs of the reputed syndrome. I will try later to state the contradictions that occur in the establishment of this syndrome. In this study such cases as have been given the diagnosis of so-called status lymphaticus show unquestionable and precise signs of internal glandular disturbance and the type, whether given the name of status lymphaticus or not, becomes quite significant. The purpose is to show the large occurrence of this type of individual among cases of war neurosis and to say that this finding adds new importance to the physiologic factors in the etiology of the war neurosis.

PHYSICAL FINDINGS IN STATUS LYMPHATICUS

The physical findings said to exist in status lymphaticus can be stated according to systems of the body. The most widespread relate

to the lymph tissue; the most debatable to the circulatory system; the most numerous affect the bones; the most conspicuous relate to the skin.

First, in regard to the skeletal frame: In status lymphaticus, it has been said that the skeleton shows undue slenderness of the long bones, a narrowing and lengthening of the thorax, prominent and winged scapulae, relative increase in the antero-posterior measurement of the skull, lordosis, a widening of the pelvis and arching of the femur. Secondly, there is presumably a hyperplasia in the lymph system with generally enlarged glands and increased lingual, tonsillar and pharyngeal lymph tissue; also, the lymph nodes of the intestinal tract are hyperplastic, the spleen enlarged, the thymus persistent and of undue size. Thirdly, in the circulatory system small caliber of the arteries, especially of the aorta, and a small heart are supposed signs of status lymphaticus. Fourthly, the skin and its hairy developments, according to the syndrome, show characteristic features. The skin in these cases is velvety and of a feminine delicacy, and the hairy growth approaches or reaches the feminine type in amount and distribution. The hair of the head is abundant, however, and only that on the body is scanty. The deficiency amounts to a very thin growth of hair on the upper lip, absence or great scantiness of hair elsewhere on the face, absence of hair on the chest or about the nipple, pubic hair scanty in amount and with a horizontal delimitation along its upper border and not ascending in the midline to or toward the umbilicus. Finally, in a different sphere congenital anomalies of the reproductive organs are said to be common. Largely as a result of the combined skeletal findings and the nature of the skin, there is often a rounding of the contour of the body so that femininity is again resembled. This is supposedly most noticeable in a rounding of the upper arm.

Haven Emerson, in 1914, published some studies on status lymphaticus. To the exclusion of many of the symptoms mentioned by former writers, he came to the conclusion that the diagnosis of status lymphaticus rests on scantiness or absence of hair on the lip, chin and cheek, scantiness of axillary hair, together with the feminine type of pubic hair, a tendency to narrowness and abnormal length in the thorax, a noticeable rounding of the thighs, a rounding of the upper arm, and a markedly velvety skin. Emerson concluded that there is no consistent parallel occurrence of such features as an increased relative length of skull, prominence of the scapulae or lordosis. These skeletal findings were not more frequent among markedly hypotrichotic cases than among cases of an opposite type. The heart and arterial changes were not found to occur in any convincing percentage, nor were the genital abnormalities found. He excluded changes in the lymph glands

for the reason that they are subject to too many extraneous influences. The spleen was not enlarged in his cases and the thymus was not tangible.

AUTHOR'S OBSERVATIONS ON TWO GROUPS OF
SOLDIERS EXAMINED

I examined cases along all the lines mentioned by the earlier writers, but I agree with Emerson that the lymph system findings are not suitable for classification one way or the other. I obtained figures to show that no circulatory or genital findings are characteristics of the condition, and that neither are a majority of the reputed skeletal signs. In virtual agreement with Emerson, the writer comes to the conclusion that the only features that are persistent and mark the type and without which the condition cannot be said to exist are: extreme scantiness of the hair of the face, trunk and extremities, a feminine type of pubic hair and a velvety skin. Emerson does not speak of an abundance of scalp hair in contrast to the hypotrichosis elsewhere, but the writer found it in a majority of the cases and believes this is a feature of the picture. In addition, there was found in the majority a tendency toward the long, narrow type of chest and, likewise, a tendency toward a slenderness of the long bones. To repeat, the writer uses the term so-called status lymphaticus to describe this restricted syndrome—bodily hypotrichosis in contrast to abundance of hair on the scalp, feminine type of pubic hair, velvety skin, a tendency toward abnormal length and narrowness of the thorax and slenderness in the long bones. And it is worth while at this point to note that in a male the signs of so-called status lymphaticus are those which constitute a partial heterosexualism.

The cases examined were two groups—one a group of psychoneurosis cases, and the other a group of surgical cases. The psychoneurosis cases, numbering 114, were seen in Ward 55 of U. S. General Hospital No. 1, and were cases of psychoneurosis that developed in American soldiers on duty in France, the huge majority of them going through trench warfare. They were unselected cases and to a large degree were merely consecutive admissions. The psychoneurosis diagnosis was made by the psychiatric staff, presided over by Major George H. Kirby. The other group of cases—119 in number—were surgical cases, all battle casualties, examined at U. S. Debarkation Hospital No. 3. The patients were soldiers who had gone through front area duty and who had not developed war neurosis. They likewise were entirely unselected. They were not consecutive as regards admission to the hospital, but were either seen in the course of examination for organic nerve injuries or taken, at random, in various wards, all the patients present in a ward being examined without

discrimination. Both groups were of enlisted men and included no officers.

The two groups, then, were of soldiers sharply contrasted, it can be claimed, in their reaction to modern warfare. Were these groups similarly in contrast when judged according to the status lymphaticus syndrome? The hypotrichotric individual with feminine type of pubic hair, with a feminine velvety skin, slenderness of the long bones and a narrowness of the thorax, was found virtually twice as frequently among the psychoneurotic cases as among the surgical cases. To give the figures — among 114 cases of psychoneurosis studied, twenty-seven were of this "status type," an occurrence of 23.68 per cent., while among 119 surgical cases studied, only fifteen showed the syndrome — an occurrence of 12.60 per cent. It would seem figures unlike to that extent must carry some significance. And what are the inferences to be drawn?

The status lymphaticus syndrome is based on endocrinal disease, and since among war neurosis cases virtually twice as many individuals showed status lymphaticus as among other soldiers, our first conclusion is that an endocrinal abnormality, in some fashion, increases susceptibility to the neurosis.

A second conclusion growing out of the first quite naturally is that in considering the manner of development of a war neurosis physiologic factors need greater emphasis. Insufficiencies of a small or large degree which are surely physiologic in their background, whether working internally only or manifested as well by palpably bad behavioristic traits, cannot be ignored. They occur, and in the introspection which is offered by at least certain phases of the war game, they become all important. Such insufficiencies can be endocrinal. They are, in that instance, endocrinal, chemical and physiologic, and in the war neurosis have a rôle.

Finally, a different phase needs consideration. An hereditary psychopathic makeup is known to be associated with the status lymphaticus syndrome in a conspicuous degree. Emerson, in 1914, contrasted the occurrence of status lymphaticus among the patients in the alcoholic and drug addiction wards of Bellevue Hospital with its occurrence in the general wards. He found the incidence of status lymphaticus among the alcoholics was 22 per cent. — four times that in the general wards, and in another study he found that among dementia praecox cases the percentage is even higher. He says: "In the general medical wards of Bellevue Hospital . . . the incidence is less than a quarter of that found among alcoholics." Further, he concludes: "They (the status cases) cannot be expected to survive as well as normal men extreme fatigue, shocks, loss of blood, abuse

of narcotic drugs or surgical proceedings." Even more plainly established, however, is an association between status lymphaticus and hereditary tendencies of a psychopathic nature. This finding is applicable in the present study; for in figures concerning the large occurrence in the war neurosis of status lymphaticus—itsself an earmark of psychopathic makeup—we have evidence that a psychopathic makeup is frequent in the war neurosis. It is confirmatory evidence of a conception that is not new and one that no one has expressed as well as Farrar, who writes: "It remains true that in the majority of severe war neurosis of all types there is evidence of a personal element of psychopathic potential."

SUMMARY

The incidence of so-called status lymphaticus in soldiers with psychoneuroses is virtually twice that in wounded soldiers who did not develop a neurosis. This finding appears to indicate that in the etiology of the war neurosis an endocrinal abnormality increases susceptibility to the neurosis. It emphasizes the fact that in many, if not in all cases, strong etiologic factors are at work in the physiologic domain without minimizing the importance of factors that are psychical. Rather, it brings new proof, of a physical character, of the conception that, in the war neurosis, an initial weakness operative in the psychical field, is essential.

The writer is deeply grateful to Major Edward Pershing, M. C., U. S. Army, and to Dr. George H. Kirby, formerly major in the U. S. Army, for permission to study the cases in their respective wards, and especially in debt to Dr. Kirby for numerous helpful suggestions.

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A STUDY OF HYSTERIA, BASED MAINLY ON CLINICAL MATERIAL OBSERVED IN THE U. S. ARMY HOSPITAL FOR WAR NEUROSES AT PLATTSBURG BARRACKS, N. Y.

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Introductory Remarks

Purely Functional and Mental Nature of Hysteria

Analysis of Two Selected Groups of Hysteria

Etiologic Mechanism of Hysteria

Acute Emotional Disorders to be Distinguished from Hysteria

Illicit Motive as Factor in Etiology

Psychic Mechanisms Underlying Cures

Particular Method of Therapy of Comparatively Little Importance

Spontaneous Cures

Circumstances Contributing to the Prevalence of Hysteria

Relationship Between Hysteria and Malingering

Distinction Between Hysteria and Malingering

Sex Factors—Intrapsychic Conflicts

The Hysterical Personality

New Designation Suggested for Hysteria

Neurasthenia

Striking Case Demonstrating the Remarkable Stability of a Normal Neuropsychic Constitution

Lessons for Civilian Practice.

Psycho-Analysis in War Neuroses

Summary

INTRODUCTORY REMARKS

It has been repeatedly observed that the apparent incidence of nervous and mental disorders in armies in times of peace is higher than in civil populations. The causes which combine to produce this general result are probably many and varied. To a certain extent, however, this statistical showing is to be ascribed not indeed to higher incidence, but to the fact that the exactions of military life bring to light constitutional weaknesses which, under other conditions, pass unobserved.

In times of war the apparent incidence is further and enormously increased, especially through the development of psychoneuroses; and thus the world war of 1914-1918 furnished experiences which brought into great relief certain neuropsychiatric problems.

War neuroses have their counterparts in peace times and in civilian life—particularly in the so-called traumatic neuroses and in hysteria. The war, however, created an unprecedented opportunity of observing such cases in large numbers and under conditions forcing into view their underlying psychic mechanisms.

The main object of this paper is to report in condensed and convenient form some of the material observed in the U. S. Army Hospital for War Neuroses at Plattsburg Barracks, N. Y., together with an analysis of it in the light, not only of my own experience, but also of published experience of others and of pre-war conceptions of psychoneuroses.

PURELY FUNCTIONAL AND MENTAL NATURE OF HYSTERIA

My experience has led but to a full confirmation of the long prevailing view of physicians that *hysteria is purely functional in its nature*. Hysterical manifestations not infrequently fasten themselves to a wound or a scar; often they are attributed by the patients to such physical factors as shell shock without wound, or to being buried in débris, becoming exhausted from continuous marching with heavy equipment, etc. An analysis of the cases, however, readily reveals the fact that *the mechanisms underlying hysterical symptoms are purely mental* and that the physical factors apparently involved operate not as such but through their psychic accompaniments, such as fright, disgust, rebellious feeling, suggestion, etc.

A striking instance of hysterical symptoms arising through suggestion originating in a trivial wound or scar is that in the case of B., Co. C., 23d Inf., who was admitted by transfer from Base Hospital No. 8, Savenay, France, on Oct. 15, 1918. He had a flat circular scar, about the size of a five-cent piece, on the outer side of the right leg. This scar marked the site of a superficial shrapnel wound sustained on the Verdun front on March 18, 1918. The fragment penetrated only the clothing and lodged in the soft tissues of the leg so superficially that another soldier was able to pry it out with the point of his penknife; iodine and bandage from a first-aid packet were applied and the patient sought no treatment by a medical officer. In about three weeks the wound healed completely. A few days later he developed "shell shock" with paralysis of both legs which eventually left a limping gait due to weakness of the right leg and an area of hypoaesthesia most marked around the scar but in a lesser degree extending from the knee to within an inch of the ankle on the outer one-third of the circumference of the leg. Shortly after the armistice the patient recovered and on Dec. 12, 1918, he was returned to duty.

The view that the mechanisms underlying hysterical symptoms are purely mental is held almost unanimously by those of experience.

Möbius,¹ M. Lewandowsky² and Imboden³ may be cited as random examples.

1. Jelliffe, S. E.: Hysteria, in *Modern Medicine*, edited by Osler and McCrae, Philadelphia, P. Blakiston's Son & Co. 5:655.

2. Lewandowsky, M.: Erfahrungen über Behandlung nervenverletzter und nervenkranker Soldaten, *Deutsch. med. Wchnschr.*, Dec. 30, 1915.

3. Imboden, K.: Das Neurosenproblem im Lichte der Kriegsneurologie, *Cor.-Bl. f. schweiz. Aerzte* 47:1098 (Aug. 25) 1917; abstr., *J. A. M. A.* 69: 1305 (Oct. 13) 1917.

Although there is almost complete unanimity on the point of hysteria being a purely mental disorder, there is considerable divergence of views as to its exact nature and as to the mechanisms underlying its manifestations. Most of the current conceptions were formulated prior to the war. A brief restatement may be permitted.

Charcot's conception was that of a disease entity. This conception led to a preoccupation with symptomatology, differential diagnosis, clinical definition, and largely remained on a descriptive level.

Möbius saw in hysteria not a disease entity but a biologic trait characterized by a special type of reaction. "For him every one was more or less hysterical. Every one has hysterical small coin in the bank of his personality."⁴

Janet's contribution consists essentially in the theory of subconscious mental processes. An idea or a group of ideas may operate somewhere beneath the threshold of consciousness. For him "Hysteria is a form of mental depression, characterized by the retraction of the field of personal consciousness and by a tendency to the disassociation and the emancipation of systems of ideas and of functions which by their synthesis constitute the personality."⁵

Further progress in the analysis of hysterical mechanisms is due to Freud.⁶ He attempts an explanation of the phenomenon of splitting or doubling of personality to which Janet has called attention. Ideas or complexes of ideas are lodged in the region of the subconscious not at random but by a purposeful functional process, which he terms *repression*, by reason of being charged with painful affect. The important part played by affect in the etiology of hysteria had, of course, long been sensed and had been insisted on in particular by Binswanger. Freud's experience has led him, moreover, to assume the universality of a *sexual origin* of the repressed complexes underlying hysterical manifestations.

The final principle of the Breuer-Freud hypothesis is the principle of *conversion*. The strangulated affect, the unreacted-to emotion, belonging to the disassociated state which has been repressed, finds its way into bodily innervation, thus producing the motor phenomena of hysteria. In this way the strong idea is weakened by being robbed of its affect—the real object of conversion.⁷

4. Jelliffe, S. E.: Hysteria, p. 656.

5. Quoted by S. E. Jelliffe (Footnote 1).

6. Freud, S.: Selected Papers on Hysteria and Other Psychoneuroses, English translation by A. A. Brill, New York, Nerv. and Ment. Dis. Pub. Co.

7. White, W. A.: Current Conceptions of Hysteria, Interstate M. J. January, 1910.

There remains to be mentioned the contribution of Babinski,⁸ which has largely dominated the French schools not only in pre-war years but even through the war, having apparently survived the light of the great mass of newly added experiences.

The essence of Babinski's contribution consists in an attempt to isolate from the heterogeneous mass of material traditionally thrown together under the heading of hysteria the elements of which it is composed. An application of more careful diagnostic technic has enabled him to eliminate organic cases, emotional disorders and reflex disorders, leaving behind a more restricted hysteria to which he has applied the newly-coined term *pithiatism*. For him *hysteria, in this restricted sense, consists in manifestations which are brought into existence by the influence of suggestion and the cure of which takes place by persuasion*.

Perhaps the most significant point insisted on by Babinski is the necessity for distinguishing true hysteria from simulation, especially where the latter manifests itself in characteristically hysterical phenomena—paralyses, contractures, anesthesia, etc. The result of treatment by persuasion here becomes the basis of the differentiation: If persuasion fails to cure, the case is not hysteria but simulation.

The question may properly be raised, *How do the facts, which have been observed in the course of our experience, square with current pre-war theories?*

A study of the Plattsburg material seems to indicate the following:

1. Hysterical phenomena are a special group of manifestations arising on a basis of neuropathic constitution.

2. Neuropathic heredity is probably the essential factor in the etiology.

3. The frequent occurrence of dissimilar heredity and, in the patients themselves, of neuropathic traits not specifically hysterical, points to a relationship of some sort between hysteria and other manifestations of neuropathic constitution.

4. The hysterical tendency varies widely in degree and shades off toward normal constitution by gradual transition. Thus, in some cases the tendency becomes manifest in childhood and continues to be so through life under ordinary conditions, while in other cases it remains latent until brought to light by some extraordinary condition on removal of which it lapses again into latency.

In other words, this material leads away from Charcot's conception of hysteria as a disease entity and rather toward that of Möbius,

8. Babinski: *Démembrement de l'hystérie traditionnelle. Pithiatisme*, *Semaine méd.*, Jan. 6, 1909; Babinski and Froment: *Hystérie, pithiatisme et troubles reflexes*, Collection Horizon, Paris, 1916.

which regards clinical hysteria merely as exaggerated manifestation of a common biologic trait. These views are based mainly on data which have been summarized in Tables 1-6.

ANALYSIS OF TWO SELECTED GROUPS OF CASES OF HYSTERIA

These data arise from two selected groups of cases of hysteria. Group 1 consists of fifty patients in whom hysteria existed prior to enlistment and was not incurred in line of duty; patients disposed of by discharge on certificate of disability. Group 2 consists of fifty patients in whom no evidence of the existence of hysterical manifestations prior to enlistment could be obtained; in these patients the condition was, therefore, considered in line of duty; all of them eventually recovered and returned to duty. Naturally, the more pronounced cases are those of Group 1.

Table 1 shows neuropathic heredity in a high percentage of cases in both groups, but in a higher percentage in Group 1 than in Group 2.

TABLE 1.—NEUROPATHIC HEREDITY

	Group 1, per Cent.	Group 2, per Cent.
Family history positive for neuropathic heredity.....	76	52
Family history negative for neuropathic heredity.....	24	46
Family history unascertained.....	—	2

Table 2 shows in detail the various neuropathic conditions recorded in the family histories of hysteria patients. It demonstrates the fact of dissimilar heredity and the greater prevalence of the various neuropathic conditions represented therein in the families of patients in Group 1 than in those of Group 2.

TABLE 2.—NEUROPATHIC CONDITIONS IN FAMILY HISTORIES
OF HYSTERIA PATIENTS

	Number of Cases	
	Group 1	Group 2
Epilepsy	14	5
Fainting spells	11	2
Sick headaches	8	6
Insanity	10	6
Feeble-mindedness	—	1
Alcoholism	7	10
Criminalism, eccentricities, temperamental anomalies.....	15	6
Other nervous diseases (nervousness, breakdown, etc.).....	42	36
Total.....	107	72

Table 3 shows social maladjustments prior to enlistment. These consist in poor progress in school, poor showing in work (frequent aimless changes of occupation, low earnings, long periods of idleness,

etc.), intemperance, criminalism, domestic maladjustments, etc. The table shows that the percentage of social maladjustment is much higher in Group 1 than in Group 2.

TABLE 3.—SOCIAL MALADJUSTMENTS PRIOR TO ENLISTMENT

	Group 1, per Cent.	Group 2, per Cent.
History positive as to social maladjustment.....	62	40
History negative as to social maladjustment.....	38	56
History unascertained	—	4

Table 4 shows in detail the various items of social maladjustment, giving, for the two groups respectively, the number of instances of each item. There are more items of maladjustment than cases of maladjustment, in both groups, as in many cases there is a record of more than one item. But it should be noted that the excess of items of maladjustment in Group 1 as compared with Group 2 is out of proportion to the excess of cases of maladjustment. We find, indeed, that while cases of maladjustment in Group 1 as compared with Group 2 are in the proportion of 155:100, items of maladjustment are as 227:100.

A further analysis of the data shows that 86 per cent. of the cases in Group 1 and 68 per cent. of those in Group 2 present either neuro-pathic heredity, or a history of social maladjustment, or both.

In other words, the data indicate that Group 1 is characterized not only by a greater percentage of cases of defective constitutional make-up, but also that the defectiveness is of a graver sort in the average cases in that group: it becomes manifest at an earlier age and is more persistent and more general.

TABLE 4.—ITEMS OF SOCIAL MALADJUSTMENT

	Number of Instances	
	Group 1	Group 2
Poor progress in school	24	9
Poor showing in work.....	13	2
Intemperance	8	9
Criminalism	4	2
Other maladjustments (domestic, etc.).....	10	4
Totals.....	59	26

Thus I find in these data an explanation for their maladjustment to the military régime and for variation in degree of such maladjustment. Turning now to the military record of these soldiers I find that the data afford a rough measure of such maladjustment, i. e., something more than a mere impression.

It will be seen from Table 5 that the great majority of the men in Group 1 did not enlist voluntarily but were drafted, while the opposite is true of the men in Group 2; also that a greater number of the men in Group 1 are known to have claimed exemption from the draft. It will be understood, of course, that in none of the cases had any claim for exemption been found valid by the draft boards.

TABLE 5.—MANNER OF ENTRANCE INTO MILITARY SERVICE

	Group 1	Group 2
Enlisted voluntarily	11	32
Drafted	39	17
Unascertained	—	1
Claimed exemption from draft.....	5	2
Did not claim exemption.....	19	37
Unascertained	26	11

The principal manifestation of hysteria is invalidism of one sort or another without organic basis. However, all the cases in the two groups under consideration were ambulant cases. Though presenting, perhaps, a limping gait, a tic, a tremor, a contracture, a paralyzed arm, aphonia, a stooped-over position, or, on occasion, fainting spells or even violent convulsions, the symptoms were disabling only as regards drilling, hiking or duty of any kind, but not as regards taking a leisurely constitutional, attending an entertainment, visiting lady friends in town, being absent without leave or eating three meals a day.

In the light of these considerations, the amount of time spent in a hospital by an hysterical patient acquires a special significance as throwing a side light on the degree of constitutional defect present in a given case.

In Table 6 the cases of both groups have been divided according to the fraction of time spent in hospitals since entering the army. It will be seen that the men in both groups spent a strikingly large part of the time in hospitals, but that those in Group 1 did so to a far greater extent than those in Group 2. The majority of the men in Group 1 rendered little or no service in the emergency, proving themselves instead but an added burden and expense to the government.

TABLE 6.—FRACTION OF TIME SPENT IN HOSPITALS SINCE ENTERING MILITARY SERVICE

	Number of Cases	
	Group 1	Group 2
Less than two-fifths.....	9	25
From two-fifths to three-fifths.....	16	13
From three-fifths to four-fifths.....	13	8
Over four-fifths	12	3
Unascertained	—	1

The data exhibited in Tables 5 and 6 afford some hint as to the motives which actuate hysterical conduct; a discussion of these motives follows later.

As further evidence of the constitutional defect underlying hysteria may be mentioned its very frequent association with other constitutional disorders, such as constitutional psychopathic states, epilepsy, alcoholism, and especially mental deficiency. As regards the latter condition alone, our psychologic department reported the results of intelligence tests as indicating that no less than 31 per cent. of the patients with hysteria were under 10 years in mental age.

ETIOLOGIC MECHANISM OF HYSTERIA

The constitutional basis of hysteria being granted, the question still remains, What are the immediate factors at work in the etiologic mechanism; or, more specifically, In what way are disabling manifestations produced and maintained?

The patients themselves, especially the more feeble-minded and ignorant among them, are apt to attribute their disabilities to trivial and inadequate causes. Good examples are:

Tonsillectomy producing aphonia; malaria producing aphonia; a cold producing aphonia; measles producing aphonia; herniotomy producing a limping gait; astasia-abasia caused by a 2-mile hike; "shell-shock" producing paralysis of both legs; stooped-over position, so-called *camptocormia*, produced by a slight blow on the groin and later aggravated by an operation for varicocele; typhoid and paratyphoid protective inoculation causing hemiplegia and hemianesthesia; sudden monoplegia with anesthesia affecting one arm, produced by "a stroke" without known cause; violent convulsions produced by attempt to obtain blood for Wassermann reaction test from vein in arm.

It is obvious that these alleged causes hardly deserve consideration; they are, at most, occasions out of which have arisen suggestions in part determining the particular symptomatology, rather than true causes. The causes assigned by medical officers are, as a rule, of a different order. In the early months of the war physical and psychic factors were mentioned more or less indiscriminately:

Cases of nervous and mental shock were found to be due sometimes to the bursting of heavy shell near the man, to his burial under earth and debris, or to the effects of noxious gases; in other cases to nervous exhaustion due to sleeplessness, fear, anxiety or other prolonged strain.⁹

The symptoms are always referable to the strain of active service. Sniping is important, the ghastly sights of carnage are still more responsible, but the intensest strain is high explosive shell fire.¹⁰

9. Turner, W. A.: Cases of Nervous and Mental Shock Observed in the Base Hospitals in France, *Brit. M. J.*, May 15, 1915.

10. Forsyth, D.: Functional Nerve Disease and the Shock of Battle, *Lancet*, Dec. 25, 1915.

Campbell names three etiologic factors in these neuroses—the noise of the exploding shells, the violent atmospheric concussion, and the fear caused by the terrifying war conditions.¹¹

Myers divides mutism into two groups according to causes—physical and psychic. His impression is that the two groups occur with equal frequency.¹²

Gradually, as the distinction became clear between true cerebral concussion and psychoneuroses, the view gained ground that physical factors, as such, played no part in the etiology of psychoneuroses.

Bonhöffer noted that many symptoms of war neuroses were such as fright itself may directly induce. Karplus found that while physical traumata were often present, they were also at times absent.¹³

The explosion has not only physical effects, but also a psychic one, which consists in an emotional shock. In certain cases this *emotional shock* dominates the situation to the point of being alone responsible for the neuro-psychic symptoms which a hasty and superficial consideration at first placed in relation to cerebral concussion; and it is because both give rise to an emotional shock that the explosion of a shell and a terrifying sight find expression in the same syndrome.¹⁴

ACUTE EMOTIONAL DISORDERS TO BE DISTINGUISHED FROM HYSTERIA

In studying the etiology of war neuroses it would seem important to distinguish the acute emotional disorders observed at the front from hysteria. This distinction has been, perhaps, most clearly drawn by Léri who had opportunities of seeing cases at the front, in field hospitals and in neuropsychiatric centers in the interior. According to this author, it is a mistake to think that hysterical manifestations are an integral and necessary part of the emotional syndrome. They can appear independently of all emotion; and the emotional syndrome has nothing in common with hysteria.

It seems that the psychic factors to which war neuroses in general are attributed—fright caused by danger from projectiles, horrifying sights, etc.—play a part only in the acute emotional syndrome; hysterical phenomena are not directly produced by them. On this point there is unanimous agreement among those who have had opportunities of observing cases at the front.

It is a remarkable fact that hysterical manifestations never appear except when a patient is in a place of security: in a trench, a dugout, a first-aid sta-

11. Campbell, H.: War Neuroses, Practitioner, London, May, 1916.

12. Myers, C. S.: Contributions to the Study of Shell Shock (IV), Lancet, Sept. 9, 1916.

13. Birnbaum, K.: Kriegsneurosen und -psychosen auf Grund der gegenwärtigen Kriegsbeobachtungen; erste Zusammenstellung vom Kriegsbeginn bis Mitte März, 1915, Ztschr. f. d. gesamte Neurologie u. Psychiatrie, 1915; reviewed by August Hoch in New York State Hosp. Bull. 8:287, 1915.

14. Rogues de Fursac, J.: Traumatic and Emotional Psychoses; So-Called Shell Shock, Transl. by A. J. Rosanoff, Am. J. Insan. 75:19 (July) 1918.

tion. Moreover, they disappear, as by magic, either when security is diminished, as by the dugout being shelled, or when the ambulance is in sight for the evacuation of the wounded. Often they appear during the period of artillery action in preparation for an attack; but never has an hysterical convulsion or paralysis been seen in the open, on the battlefield, under exposure to a barrage—a fact which seems to us of major interest for the understanding of the pathogenesis of hysterical disorders.¹⁵

Psychoneurotic affections need not follow directly on the causative emotional disturbance, but an incubation period of varying length may intervene. In the firing line psychoneuroses are rare. But once he is out of danger, the psychopath, no longer dominated by instinct, is a prey to any abnormal mental and neurotic states that conditions or events, present or previous, may arouse in him. These manifest themselves in contractures, paralyses, tremors, convulsive fits, etc.¹⁶

Those disorders which develop at the front and are the direct expression of violent emotion are never of long duration; they last scarcely more than a day or two; the intensity of their manifestations subsides rapidly and almost all patients can be returned to the front within a few days. Many, it is true, have to be kept in the field hospital as long as a week or ten days, but this is generally due not to persistence of the emotional disorder proper, but to the physical exhaustion from overexposure, overexertion, lack of sleep, lack of food, etc., which usually accompanies it.

Capt. William Brown reports that of 200 nervous and mental cases which passed through his hands in December, 1916, 34 per cent. were evacuated to the base after seven days' treatment, and 66 per cent. returned to duty on the firing line after the same average period of treatment.¹⁷

The immense majority of cases of emotional disorder return to the front, cured, without ever reaching the zone of the interior.¹⁸

The recovery in cases of uncomplicated emotional disorder is usually complete. Of course a tendency toward recurrency remains. In some cases it can be demonstrated, according to some observers, that a heightened emotional susceptibility has been produced.

To becoming accustomed to emotions, which fortunately is the rule, there is opposed a certain "anaphylaxis" toward emotions; but in the genesis of such anaphylaxis, it should be stated, one is obliged to go back to a certain degree of predisposition which is more or less constitutional.¹⁹

15. Léri, A.: *Commotions et émotions de Guerre*, Paris, 1918; reviewed by A. J. Rosanoff in *Arch. Neurol. & Psychiat.* 1:273 (Jan.) 1919.

16. Molinari, G.: *Nosografia delle psiconeurosi di guerra*, *Riforma med.*, Naples 33:898 (Sept. 15) 1917.

17. Salmon, T. W.: *The Care and Treatment of Mental Diseases and War Neuroses ("Shell Shock") in the British Army*, New York, The National Committee for Mental Hygiene, 1917.

18. Léri, A.: *Commotions et émotions de Guerre*, Paris, 1919; reviewed in *Arch. Neurol. & Psychiat.* 1:273 (Jan.) 1919.

19. Léri, A.: *Commotions et Emotions de Guerre*, Paris, 1918.

The psychoneurotic symptoms which persist and which force the evacuation of cases to the rear (save possibly those seen in the rare cases of "emotional anaphylaxis"), and more particularly hysterical symptoms, are, thus, evidently to be regarded not as direct products of emotional shock but as arising, after the lapse of a greater or lesser interval of time, by a different psychic mechanism.

ILLICIT MOTIVE AS FACTOR IN ETIOLOGY

I come now to the consideration of a factor which, though seldom frankly avowed by patients and seldom definitely established by clinical investigation in individual cases of hysteria, yet has made itself clearly apparent to many observers in all armies. Speaking for myself and with special reference to my experience in this hospital, I would say that this factor has obtruded itself on my attention until I have come to regard it as the mainspring of hysterical conduct.

This factor consists in a *concealed, illicit, morally untenable motive*.

The motive is not always the same, but it is always characterized by the above mentioned qualities. Its most frequent variations are:

1. To evade the law of conscription.
2. To procure, on reporting for physical examination at a training camp, rejection for physical unfitness.
3. To evade dangerous, disagreeable or difficult duty, or to evade all duty.
4. To procure the ease and privileges of hospital care.
5. To procure discharge on certificate of disability.
6. To procure compensation for disability.

As stated above, not I alone, but also other observers, in all armies, have been impressed with this factor in the mental mechanism of hysteria and have more or less clearly formulated their impression.

The psychological basis of the war neuroses (like that of the neuroses in civil life) is an elaboration, with endless variations, of one central theme: escape from an intolerable situation in real life to one made tolerable by the neurosis.²⁰

As long as war is in progress and return to the front is imminent the patient prefers (unconsciously at least) to retain his disability rather than to face the perils and discomforts of trench life.²¹

Speaking in particular of refractory hysterical phenomena, Léri says: "In all cases they originate not in emotion, but in reflection, when emotion no longer exists; they originate in the idea of retirement on pension with its double benefit of further removal from danger and pecuniary indemnity."²²

20. Salmon, T. W.: The Care and Treatment of Mental Diseases and War Neuroses ("Shell Shock") in the British Army, New York, The National Committee for Mental Hygiene, 1917.

21. MacCurdy, J. T.: War Neuroses, Psychiat. Bull., July, 1917.

22. Léri, A.: Commotions et émotions de Guerre, Paris, 1918.

The "wish" is certainly a strong etiologic factor in these functional conditions. This is shown by the fact that, with injuries which *a priori* render a soldier unfit for further active service, they are very rarely found.²³

The motivation of psychogenic neuroses consists of a subconscious wish to escape the events which produced them—a "recourse to neurosis," at least in so far as the picture is dominated by hysteria and whenever there has been an internal conflict. In fright neuroses there is no such motivation.²⁴

Henneberg does not expect much from psychotherapy for soldiers who have been at the front. The men have no wish to recover and be sent back.²⁵

In the individual case direct evidence of illicit motive is seldom available. This, however, is in the nature of things and is not surprising. Yet not infrequently patients make unguarded remarks which amount almost to a confession. These are some examples:

1. A soldier was asked, How old were you when you were drafted? "I was within ten days of being 32 years of age. They beat me to it by ten days." (H., Hqs. Co., Classification Camp, A. E. F., France.)

2. Q. If you were as much disabled when you were drafted as you are now, why did you not claim exemption? A. "It was no use. Our local board took in everybody regardless. You'd have to be dead to get exemption." (P., 51 Co., 159 Depot Brigade, Camp Taylor, Ky.)

3. A soldier had a violent hysterical convulsion on the veranda in front of the window of the office of the senior member of the disability board. After about twenty minutes of violent commotion, he arose and came, weeping, into the office: "Oh, Major, did you see me have that spell?" Yes, I did. "Oh, Major, don't give me an S. C. D." (Surgeon's certificate of disability for discharge). I am afraid I shall have to. "Very well, sir, it's up to you; you are the head of the board." (F., 2 Tank Co., A. E. F., France.)

4. One soldier simply declared, "I am a case of constitutional inferiority; a good many doctors said that; the local board had no business to draft me into the army; it is the duty of the medical officers to rectify the mistake; it is their duty to discharge me." (M., 50 Co., 159 Depot Brigade, Camp Taylor, Ky.)

The avowed intention was almost always to serve the country and even die for it; but "Captain ——— in Base Hospital ——— told me I was not fit for the army. If I am not fit for duty, then why don't you give me my discharge?" This argument, in one form or another, was very common among those whose motive it was to secure discharge on certificate of disability.

That illicit motive, and it alone, and not shell concussion, war strain, emotional shock, etc., is the factor which actuates hysterical conduct is further shown by three groups of observations.

23. Lewandowsky, M.: Erfahrungen über Behandlung nervenverletzer und nervenkranker Soldaten, Deutsch. med. Wchnschr., Dec. 30, 1915.

24. Saaler, B.: Ueber nervöse und psychische Krankheiten in ihren Beziehungen zum Kriegsdienst, Berl. klin. Wchnschr., Dec. 25, 1916.

25. Zeehandelaar, I.: Nederl. Tijdschr. v. Geneesk. 2:41 (July 1) 1916; abstr., J. A. M. A. 67:890 (Sept. 16) 1916.

In the first place are to be mentioned the cases of hysteria arising in training camps in this country. In every National Army cantonment many cases came to light often on the day of reporting for military duty, more often early in the course of training—at any rate before any “war strain” could possibly have made itself felt. These cases presented all the manifestations of hysteria which, when seen in overseas cases, have so often been attributed to “shell shock.”

In the second place is the striking fact that among prisoners of war who have been, like other soldiers, exposed to shell fire, strain, etc., scarcely any cases of hysteria or other psychoneuroses have been observed.

Mörchen found only five cases of war neuroses among the 40,000 war prisoners at the Darmstadt prisoners' camp. There is every reason to assume that they had been subjected to the same kind of mechanical and emotional stress as the German soldiers, among whom hysteric nervous disturbances were so common. Lust,²⁶ reviewing 20,000 war prisoners, found the war neuroses rare among both military and civilian persons.

Victims of war neuroses usually are not taken from among the wounded, contrary to Oppenheim's statement. They seldom occur among prisoners even when they have been shocked severely. Prisoners are actuated by one great desire; to remain well so that when they are exchanged they may return home in good health.²⁷

In the third place is the very common experience of quick and complete recovery from hysterical symptoms on evacuation to a base hospital, followed by a return of the symptoms in the same or even a greater degree of intensity or by development of new symptoms on any prospect arising of again being sent to duty. Many of the overseas patients have furnished such a history. The relapse has generally occurred either on the way from the hospital to a reclassification camp or shortly after arrival there. Sometimes no cause is given for the relapse; at other times a trivial cause.

1. “The very day I came to the replacement camp from the nerve hospital, I was made so nervous by the men shouting and knocking their mess kits against the wall that I could not speak my name and shook all over.” (B., Co. C, 23 Inf.)

2. “I was sent from Base Hospital No. 34 to the replacement camp and on the following day assigned to duty. That evening as I stood on a barrel looking in through the window at a Y. M. C. A. entertainment, a gasoline engine a short distance away made a sharp noise. This frightened me greatly; I began to shake violently and got off the barrel on which I was standing.” He was ordered to remain in quarters by a medical officer; the following day he was no better and remained in bed; on the day after, still being no better,

26. Lust, F.: *Kriegsneurosen und -gefangene*, München. med. Wchnschr. 68:1829 (Dec. 26) 1916; abstr., J. A. M. A. 68:667 (Feb. 24) 1917.

27. Gaupp, R.: J. A. M. A. 68:647 (Feb. 24) 1917.

he had to be readmitted in Camp Hospital No. 26. After two weeks in the hospital his nervousness continued and he was transferred to Base Hospital No. 8, thence to be returned to the United States (M., Co. C, 28 Inf.).

Others have recorded similar observations.

Severe types that exhibit well-marked symptoms after six months do not recover in the army. A considerable proportion of those who "recover" in hospital break down again at their depots or command depots.²⁸

In the case of very slight affections, to the casual observer the patients seemed well and ready for action. But the whole state of affairs was altered as soon as the physician suggested that they begin active service again. Reassuring suggestions, that had hitherto been very effective, were no longer of any avail. The patient leaves the hospital at once; at the railroad station acute symptoms appear; and, on arriving at his regiment, nothing remains to be done but return him to the hospital. In some patients, the acute symptoms reappear even before they start to rejoin their regiments. In all such cases, there seems to be the one predominating etiologic factor—fear and horror at the thought of returning to the front.²⁹

The extremely logical and extremely brutal method introduced by Kaufmann, consisting in strong electric shocks with loud staccato military commands to do certain exercises, has realized some surprising cures, but time has shown that relapses are liable to follow on the slightest provocation.³⁰

If it is true, as stated above, that the essential element of etiology in hysteria, aside from inborn predisposition, is the existence of a concealed, illicit, morally untenable motive, it is not difficult to explain why the war has produced so many cases: It created situations in which men's natures have suddenly come to be subjected to the acid test of demand for greater personal sacrifice. It was inevitable that natures deficient in physical stamina, courage and moral inspiration should become actuated by ignoble motives.

Although it has been said that special conditions of modern warfare are responsible for the great number of psychoneuroses and that in previous wars no such effects had been observed, the fact is that, under other names, in various forms, invalidism without organic basis undoubtedly existed in abundance in all wars.

It was early discovered that when men left the field for medical treatment, they were lost for the campaign, if not for the war. The aggregate absent from the two armies (on the Potomac and in Washington), which should have been united at Antietam, was 101,756. The total in action was but 87,164. Many of those absent by authority are those who have got off either sick or wounded, or under pretense of sickness or wounds, and having origi-

28. Ballard, E. F.: Some Notes on Battle Psychoneuroses, *J. Ment. Sc.*, July, 1917.

29. Gaupp, R.: *Hysterie und Kriegsdienst*, München med. Wchnschr., March 16, 1915.

30. Imboden, K.: Das Neurosenproblem im Lichte der Kriegsneurologie, *Cor.-Bl. f. Schweiz. Aerzte* 47:1098 (Aug. 25) 1917; abstr. *J. A. M. A.* 69: 1305 (Oct. 13) 1917.

nally pretext of authority are still reported absent by authority. (From General McClellan's letter to the President under date of July 15, 1862): "The reduced condition of the old regiments . . . points to the necessity of earnest endeavor to collect all the absent officers and men belonging to these organizations. I suggest that every hospital . . . be inspected within the month of October by, if necessary, scores of officers detailed for the purpose, to ferret out the old soldiers hidden away therein. Such an inspection would produce more fruit in one week than the recruiting service can in three months. And finally, I would suggest to the War Department . . . the arrest of deserters. Convalescent soldiers leave hospitals, and have done so for the past year, and return home habitually. It is the experience of every army commander that not more than a tenth of the soldiers who are left behind sick ever rejoin." (From General McClellan's letter to the President under date of Sept. 28, 1862): "Malingerers soon become adepts in feigning all manner of diseases. By June, 1862, impositions became so frequent, that discharges for rheumatism had to be prohibited in orders. Any soldier who had once returned to his village, or his home, could at any moment procure his discharge, provided a physician "in good standing" would honestly or corruptly sign a certificate. The number of men discharged in 1862, by means of the combined agencies referred to, approximated 100,000 men. The immediate effect of those discharges was to reduce all the regiments in the field to mere skeletons; their permanent effect can be seen today in the system of pensions. The total amount paid in 1903 for pensions and the expense of maintenance during the fiscal year was \$141,752,870.50."³¹

The above abridged quotations, all of which pertain to the Union Army in the Civil War, show that what we now call war neuroses were not only present then but that they constituted an evil of even greater magnitude, relatively, than in the world war of 1914-1918.

PSYCHIC MECHANISMS UNDERLYING CURES

The point of view advanced here with reference to the mechanism of hysteria is borne out not only by the conditions under which the disabling symptoms arise, but also by the conditions under which they disappear.

The factors to which patients themselves attribute their cures are apt to be, like the causes they assign for their troubles, trivial and inadequate. The following are typical examples:

1. "I was cured by a miracle of the Lord." (D., 72d Co., 159 Depot Brigade, Camp Taylor, Ky. Case of hemiplegia and hemianesthesia.)
2. "Captain N. lifted up a little bone in my ear that had got out of place and from that moment I could hear as good as ever." (S., Hqs. Co. 39 Inf. Deafness was complete and bilateral; manipulation was limited to left ear.)
3. "Captain K. gave me electrical treatment, and after the second treatment I was all cured up." (N., Co. E, 155 Inf. Labored gait due to contractures at both knees in position of partial flexion.)
4. "Captain H. cured me in less than five minutes with hypnotism." (T., Co. I, 372 Inf. Case of tremor of head, trunk and upper and lower extremities,

31. Upton, Emory: *The Military Policy of the United States*, Ed. 4, Washington, D. C., 1917.

grotesque gait, high-pitched voice, explosive utterance, tendency to repeat each word or phrase in conversation three or four times rapidly.)

5. "I ate at first one half of an apple; on offering the other half to a patient friend, he refusing it, I said right out, surprising myself, 'I will eat it myself.' I think the apple and the massage did it; therefore I will eat apples forever, they being my lucky fruit." (W., Co. D, 310 Lab. Bn. Case of aphonia.)

6. "In the hospital in France I lay helpless in bed, unable to move or even sit up. The ward surgeon said I might never be able to get up and walk again. Then I prayed the most earnest prayer I ever prayed in my life. On the following day I got out of bed, just tumbled out; from then on grew gradually better; at first was able to crawl around on the floor, then walk about on crutches. Started for the United States on July 1. On board ship used two canes in walking instead of crutches. At the Embarkation Hospital in Newport News, Va., I no longer used crutches or canes and have used none since." (S., T-7, 23 Eng.)

Turning now to the attitude of medical officers regarding cures of hysterical manifestations, I find a striking general tendency to ignore mental mechanisms underlying pathogenesis and course. Only external aspects of hysteria as a rule receive consideration. The preoccupation seems to be with description of manifestations, charting of areas of anesthesia, differentiation from various organic affections, etc. The first object is generally diagnosis; when that is established no further investigation of mental mechanism is attempted. The next step is treatment—again without reference to the mental situation; and cures are attributed to this or that therapeutic method, every method having its advocates and opponents:

Work and interest in extraneous life are the best cure for such cases.³² In the way of treatment complete physical and mental rest are essential for both neurasthenia and shell shock.³³

Massage and electricity should be avoided in hysterical cases.³⁴ That this (faradic current) is an indirect method of suggestion no one can deny; but the important point is that the procedure is efficacious, uniformly efficacious, quickly efficacious. It need only be tried to demonstrate its almost infallible action.³⁵

The results of this treatment (chloroform anesthesia combined with suggestion) have been so satisfactory that I desire to give some account of the details of the same in this brief note.³⁶ The author disapproves of the use of an anesthetic for curing deaf-mutism.³⁷

32. Turner, W. A.: Cases of Nervous and Mental Shock Observed in the Base Hospitals in France, *Brit. M. J.*, May 15, 1915.

33. Hurst, A. F.: Functional Nervous Disorders, Medical Diseases of the War, London and New York, Longmans, Greene & Co., 1917.

34. Campbell, H.: War Neuroses, *Practitioner* 96:501 (May) 1916.

35. Chavigny, P.: *Psychiatrie aux armées; thérapeutique et médecine légale*, Paris méd., Jan. 1, 1916.

36. Milligan, E. T. C.: Method of Treatment of Shell Shock, *Brit. M. J.* 2:73 (July 15) 1916.

37. Russell, C. K.: Study of Certain Psychogenetic Conditions among Soldiers, *Canad. M. A. J.*, August, 1917.

The treatment par excellence is hypnotic suggestion.³⁸ Nonne calls attention to the advantages of hypnotic suggestion. Cures are frequent and rapid.³⁹ As to hypnotism, no more need be said than that its use is prohibited in military hospitals.⁴⁰ Psychic treatment without hypnosis is recommended.⁴¹ It is striking to note how frequently hypnosis is condemned as inadequate or actually deleterious.⁴²

If further treatment is required, psycho-analysis is necessary, and the problem now becomes an everyday neurosis, the incidents of the war merely being the most recent in a life-time of mental strains.⁴³ However helpful psycho-analysis may be in time of peace, in time of war such methods appear unnecessary and undesirable.⁴⁴

PARTICULAR METHOD OF THERAPY OF COMPARATIVELY LITTLE IMPORTANCE

I am able to report from my own experience that the particular method of therapy employed is a matter of comparatively little importance. The mechanism of cure readily reveals itself when studied in the light of the above discussed mechanism of etiology. One or more of the following factors are frequently seen to be operative in cures:

1. An attitude on the part of medical officers impressing patients in such a way as to preclude any hope of successful imposition.
2. Demonstration of the unreal nature of the disability.
3. Strict discipline as opposed to sympathy, doddling or humoring.
4. Painful or otherwise disagreeable features of treatment.
5. Removal of motive actuating the symptoms by change in situation.

These factors deserve somewhat detailed discussion. The first mentioned factor is all important for the prevention of hysterical manifestations and for their suppression in the earliest stages. The frequency of hysterical manifestations arising at the front has been observed to vary extremely according to the local atmosphere created by medical officers.

For instance, in the army in which we had the direction of the neurological center, we have had the satisfaction of seeing the number of "nervous seizures"

38. Eder, M. D.: *War Shock; the Psychoneuroses in War; Psychology and Treatment*, Philadelphia, P. Blakiston's Son & Co., 1917.

39. Berlin Letter, J. A. M. A. 66:440 (Feb. 5) 1916.

40. Chavigny, P.: *Psychiatrie aux armées; thérapeutique et médecine légale*, Paris méd., Jan. 1, 1916.

41. Babinoff, Y. K.: *Injuries to the Nervous System Caused by the War*, Russk. Vrach, Petrograd, 1915.

42. *Functional Psychic Disturbances in the Light of the War*, editorial, Med. Rec., Aug. 26, 1916.

43. Forsyth, D.: *Functional Nerve Disease and the Shock of Battle*, Lancet, Dec. 25, 1915.

44. *War and Psychoanalysis*, editorial, New York M. J., Dec. 23, 1916.

diminish from the time the rumor was spread—through soldiers who had returned to their organizations without permission—that with us “the seizures were not in good repute and were regarded rather with disfavor.” As to mutisms and deaf-mutisms, we have seen them arise in certain armies in the form of veritable little epidemics, whereas in others they were almost unknown.⁴⁵

As to the second factor—demonstration of the unreal nature of the disability—it is plainly operative in the cures of aphonia and mutism by means of general anesthesia and by the “ball method,” and in the cures of paralyses, especially of the lower extremities, by means of strong electrical stimulation. Patients dread being detected in the act of simulation and therefore yield to the “treatment” rather than allow it to appear too clearly that they are able to phonate, talk or move their limbs, and yet will not do so. Under the circumstances, to fall in with the purpose of the doctors and declare themselves cured becomes for the patients the best way out of an extremely awkward and even dangerous situation.

The administration of ether for the cure of functional deafness and functional loss of voice has always in our hands proved satisfactory. It is essential that the loss of consciousness be slight, and that the patient be suddenly roused so as to realize that he is speaking or hearing.⁴⁶

A number of interesting case histories are cited to show the use of the “ball method” for curing aphonia. This method consists in introducing a ball into the larynx to cause temporary suffocation and thus produce a reflex restoring functioning power to the adductors. The method should be applied as soon as the patient has recuperated from the shock that caused the aphonia. Usually one short sitting only is necessary. The author feels that some cases of this kind are led to prolong their trouble by the desire to get as much war indemnity as possible.⁴⁷

The principle involved in the cures of paralyses and anesthetics by electricity likewise consists in surprising or forcing the patient into betraying by an involuntary movement the functional integrity of both motion and sensation in the affected limb.

The third factor—strict discipline—is unanimously insisted on by all who have had cases of war hysteria to treat, although they have so widely disagreed on other points. This factor is related to the first and supplements it by impressing on patients not only the hopelessness of imposing deception on medical officers, but also the certainty of every attempt to derive advantage from being “sick” being promptly met by measures of discipline.

45. Léri, A.: *Commotions et Émotions de Guerre*, Paris, 1918.

46. Bruce, A. N.: *The Treatment of Functional Blindness and Functional Loss of Voice*, *Rev. of Neurol. & Psychiat.*, May, 1916.

47. Muck, O.: *Weitere Heilungen von Stimmverlust im Kriege*, *München. med. Wchnschr.* 63:1466 (Oct. 10) 1916.

The fourth factor—painful or otherwise disagreeable features of treatment—very often succeeds where other methods have failed. It would seem hardly necessary to go into discussion of the mental mechanism of this factor; obviously it works by directly combating the motive by which the symptoms are produced—that of securing relief from dangers and hardships—and by substituting a new motive and a more immediately urgent one—that of getting “cured” and thereby escaping the painful treatment.

F. Kaufmann* is the accredited author of a system of treatment which even some of his fellow countrymen have considered inhuman. . . . Kaufmann worked out a system of combined suggestion and painful electrical shock, aiming at a drastic cure in one sitting. The germ of this principle dated from 1903, when, as an assistant to Erb, he observed the beneficial effects of remorseless electrical treatment in a case of hysterical muscular contraction. His system includes the four following principal features:

1. Preliminary suggestion, consisting not only of the stock methods of suggestion, but also the “atmosphere” of a hospital in which successful cures are numerous.

2. Powerful electrical shocks supplemented by vigorous “word suggestion.” As a rule, the sinusoidal current of a pantostat was preferred to the faradic current. It was combined with the galvanic current when there were symptoms of hysterical anesthesia. Some idea of the patients’ sensations may be gathered from the term *Ueberrumpelung* (unexpected attack) used in the author’s description.

3. Maintenance of an atmosphere of strict military discipline.

4. Masterfulness and pertinacity were essential; it might take hours before the desired effect was obtained, but with perseverance and the exertion of the physician’s whole personality success was ultimately achieved. . . . After the ordeal the patient should be kept in hospital a few weeks to diminish chances of relapse.

Prof. Otto Schultze** admits that this system has been stigmatized as inhuman, but does not, he thinks, inflict pain very different from that of a woman in labor. At his hospital he found Kaufmann’s system vastly superior to others.⁵⁰

Similar success has been attained in the treatment of refractory cases of hysteria by some neurologists in France by a method of painful electric shock, so-called “*méthode du torpillage*” (Vincent, Rimbaud, Trenel).

Myers found that in many cases these sensory disorders clear up spontaneously. Sometimes he saw marked improvement during an examination (a hypalgesia and hypesthesia completely disappeared after several deep pricks which the patient felt).⁵¹

48. Kaufmann, F.: München. med. Wchnschr., May 30, 1916.

49. Schultze, O.: Münch. med. Wchnschr., 63:1349 (Sept. 19) 1916.

50. Disciplinary Treatment of Shell Shock (Notes from German and Austrian Journals), Brit. M. J., Dec. 23, 1916.

51. Myers, C. S.: Contributions to the Study of Shell Shock, Lancet, Feb. 13, 1915; Jan. 8, March 18, and Sept. 6, 1916; abstr., Psychiat. Bull., July, 1917.

Some patients recover from their symptoms when the surgeons decide to undertake an operation. Some recover on being "bullied," threatened with courtmartial (S.; 4th Co., B. S., S. C.), etc. "A few recovered their speech spontaneously, and a few did so after being harshly reprimanded and isolated from other patients."⁵²

On the introduction of Wassermann tests as a routine procedure for all cases, many of our hysterical patients objected, and finally resorted to violent seizures in the laboratory. They created great disturbance, knocked the furniture about, broke glassware, and had to be held by several men. The situation was quickly controlled by the use of a whiff of ammonia "to revive" them. The patients claimed to have lost consciousness during the seizures; yet seizures have been seen to stop more than once when the chief of laboratory merely called out loudly, "Sergeant, bring the ammonia bottle!"

SPONTANEOUS CURES

Perhaps the most significant factor in the mechanism of cures is the one last mentioned in the enumeration—that of removal of motive actuating the symptoms by change in situation.

The patients which were sent to the Plattsburg hospital from overseas were for the most part those which had proved most refractory to treatment in hospital in France. Yet a great many of them had recovered spontaneously at the time they reached Plattsburg; and there was an outbreak of spontaneous cures following the signing of the armistice. Physicians who had succeeded in gaining a real insight into the mechanism of war hysteria had forseen this outcome long before it happened. The following was written in 1915:

There exists at the present time a factor by which the prognosis of emotional war psychoses is radically biased: it is the war itself. The prospect of returning to the front constitutes, for many of the emotionally unstable, an obstacle to recovery of such power that it is impossible to say if it alone might not suffice to keep up the neuro-psychic symptoms and to impart to the disease the appearance of chronicity which has been noted in some cases. The fate of these patients will not be settled until peace has been signed.⁵³

CIRCUMSTANCES CONTRIBUTING TO PREVALENCE OF HYSTERIA

That a desire to evade dangerous or difficult duty or all military duty should be prevalent in time of war is not surprising; but that it should so frequently lead to hysterical manifestations, i. e., invalidism without organic basis, requires some explanation.

52. Myers, C. S.: Contributions to the Study of Shell Shock.

53. Rogues de Fursac, J.: Traumatic and Emotional Psychoses. So-called Shell Shock, *Am. J. Insan.*, July, 1918. (English translation by A. J. Rosanoff.)

In this connection should be mentioned first of all the well-known gullibility of the medical profession. Draft evaders seem to know that they need but to complain of a disability to gain the support of duly authorized medical officers. While to laymen the scheme of these patients is often clearly apparent, the tendency of medical men is to overlook or ignore underlying motives and mental mechanisms and to allow themselves to be imposed on. Far-fetched theories are propounded even by leaders in the profession. The following examples will suffice:

Dr. Grasset suggests as an explanation of the psychology of deaf-mutism that these patients probably think that they have died, as the only thing which unites such a patient to existence is the preservation of his sensibility and sense of movement.⁵⁴

I believe this mutism is due primarily to an inhibitory functional paralysis of the voluntary cerebral nervous centers which control the management of the breath and direct its mode of escape through the glottis, mouth and nostrils.⁵⁵

The disturbance affects the highest cortical levels, the middle levels with the subconscious mechanisms for everyday activities, the motor centers in the cord with their issue in the final common path, and the muscles themselves, and often also the afferent paths and the receptive apparatus for localization and the components of deep sensibility. . . . The disorder of voluntary movement may be explained by an overaction of the cerebellum, or by want of counteraction of the cerebellum owing to the impulse from the cerebrum being in abeyance.⁵⁶

Continuous suppression or re-suppression, attended by continuous overstimulation of the instinct to action, brings about a stage of chronic hyperexcitability, resulting in physiological over-activity of the censor.⁵⁷

May not paralysis, contracture, tremor, anesthesia, etc., be due to inhibition, or to exhaustion caused by excessive stimulation of the sense organs or nervous system, or to a molecular disturbance of some kind produced by vibrations in the equilibrium of the nerve cells?⁵⁸

A double origin is postulable—a central and a peripheral genetic modus, i. e., psychic concussion and injury of the peripheral nerves. A violent impulse from external stimuli causes a functional disturbance of the delicate mechanism of the psychic centers, shown in (1) faulty distribution of motor impulses,

54. Grasset, Joseph: *Clinical Lecture on the Psychoneuroses of War*, Med. Press and Circ., June 9 and 16, 1915; abstr. by H. Butts in *U. S. Naval Med. Bull.*, January, 1916.

55. Mott, F. W.: *Psychic Mechanism of the Voice in Relation to the Emotions*, *Brit. M. J.*, Dec. 11, 1915.

56. Clarke, J. M.: *Some Neuroses of the War*, *Bristol Med.-Chir. J.*, July, 1916; abstr. by H. J. Norman, *J. Ment. Sc.*, January, 1917.

57. Ballard, E. F.: *Some Notes on Battle Psychoneuroses*, *J. Ment. Sc.*, July, 1917.

58. Sollier, P., and Chartier, M.: *La commotion par explosifs et ses conséquences sure le système nerveux*, *Paris méd.*, Oct. 23, 1915.

(2) hypo-innervation, (3) hyper-innervation, causing tremors, tonic and clonic spasms, etc., instead of single muscle actions."

The patient was a soldier who in August, 1916, became mute after the explosion of a grenade close to him. He was kept for forty days in a military hospital where various therapeutic measures were tried without success. He was discharged while still dumb, with ninety days' convalescence leave. He was arrested as a deserter perhaps because, when still suffering from the effect of the shock, he had left his regiment, and was condemned to imprisonment. He was sent to another hospital and on admission he was completely speechless, but his hearing was normal. He was placed under anesthesia and recovered speech; at first it was monosyllabic, but after a course of vocal exercises it became normal. In answering questions he stammered a little—a defect he had not had before his injury. That he was not a malingerer was shown by a certificate from the principal officer of the hospital where he had first been under observation that the condition was caused by an injury received in the service. . . . Chiadini said he could not believe that through the elective action of chloroform and ether on the lipid substances of the brain, that organ suddenly passed from a state of inhibition to a normal extrinsication of thought with correct expression in words. . . . In all cases he believed that the centers of motor and sensory speech and their relation one with the other and with the periphery were intact; but one of the two centers was separated from the ideative center, and therefore from the whole of the remaining cortex; there was therefore an interruption of the association fibers going from the whole cortex to each of the speech centers."

Undoubtedly the situation which makes it so easy for any shirker to evade duty by developing functional disability is not entirely due to gullibility of the medical profession. It is partly due to the difficult position in which medical officers are placed by military law.

It is one thing to convince oneself that in a given case there is no organic disease and only functional disability motivated by desire to evade duty or gain government compensation, and it is another thing to furnish such legal evidence as would convict a soldier of malingering in a court of law. Yet a medical officer has no other alternative than that of either designating the disability by some respectable name, such as "hysteria" or "psychoneurosis," or making a diagnosis of malingering and thereby being placed in a position of having to prove criminal intent on the part of the soldier.

The result is that, while every medical officer in the army has witnessed cases of malingering, the diagnosis of malingering is scarcely ever made. The present writer, for instance, during twenty months' service in Camp Upton and Plattsburg, witnessed many dozens of cases that he and other officers regarded as cases of deliberately

59. Oppenheim, H.: *Der Krieg und die Traumatischen Neurosen*, Berl. Wehnschr., March 15, 1915.

60. Pansera, Giuseppe, and Chiadini: *Un caso di mutismo da shock nervoso*, Policlinico, Rome, Dec. 10, 1916.

feigned disability, yet in only one of them—to be described farther on—was an official diagnosis of malingering made, it having proved possible to get conviction on that charge before General Court Martial.

RELATIONSHIP BETWEEN HYSTERIA AND MALINGERING

This brings us to the question of the relationship between hysteria and malingering. Here it should be noted that opinions differ as to when a diagnosis of hysteria should be made and when one of malingering. Yet even those who hold diametrically opposite views are agreed that there is a close similarity in the clinical manifestations of the two conditions and great difficulty in establishing the differentiation in practice. The quotations which follow are from writers both on hysteria and malingering.

Quite the most difficult problem, however, is to differentiate a conversion hysteria from malingering.⁶¹

Cases of malingering are by no means always easy to diagnose, for there is no sharp dividing line between downright malingering, mere exaggeration, subconscious malingering, and actual disease; each class merges into the next.⁶²

A most important function to be performed by a psychiatric service is the detection of true malingerers as distinguished from those soldiers who, because of psychopathic or neurotic conditions, may show almost identical indications and yet not be guilty of simulation.⁶³

The subconscious influence of desires and wishes can often be demonstrated. The pathologic fraud, closely related to the hysteric, is sometimes found, and it is easy to understand why he appears more frequently in times of war than of peace.⁶⁴

This multiform neurosis (hysteria) is not chosen by malingerers, but, more than any other disease, it renders the diagnosis of malingering extremely complicated and difficult.⁶⁵

Appreciating the fact that the diagnosis and subsequent treatment of hysteria, neurasthenia, and malingering is made difficult by the merging of symptoms common to any two or all three of the conditions, the author cites illustrative cases giving the psychology of each in an attempt to clear the atmosphere.⁶⁶

Nothing resembles malingering more than hysteria; nothing hysteria more than malingering. . . . We may examine an hysterical person and a malingerer—using exactly the same tests—and get precisely the same results in one case as in the other. The finer the methods that we employ to test the genuineness of their complaints, the reality of their objective phenomena, the

61. MacCurdy, J. T.: War Neuroses, *Psychiat. Bull.*, July, 1917.

62. Campbell, H.: War Neuroses, *Practitioner*, London, May, 1916.

63. Chavigny, P.: *Psychiatrie et médecine légale aux armées*, Paris méd., July 17, 1915.

64. Bonhöffer, K.: Erfahrungen über Epilepsie und Verwandtes im Feldzuge, *Monatschr. f. Psychiat. u. Neurol.*, July-August, 1915.

65. Bailey, Pearce: Malingering in U. S. Troops, *Mil. Surgeon*, March and April, 1918.

66. Lumsden, T.: Psychology of Malingering and Functional Neuroses in Peace and War, *Lancet* 2:860 (Nov. 18) 1916; abstr. by D. G. Sutton, *U. S. Nav. M. Bull.*, April, 1917.

more do they—in hysterical individuals—yield results which in a non-hysterical person would be held as proof of positive deceit.⁶⁷

The motives which I have found as constituting the mainspring of hysterical conduct are the same as those which students of malingering have uniformly reported as actuating their cases. Thus, Colonel Bailey⁶⁸ mentions, among others, the following motives in malingering: (a) to evade service, (b) to avoid duty, (c) to obtain exemption from punishment, (d) to obtain transfers.

Colonel Ford writes as follows: "Malingering is practiced to avoid unpleasant or dangerous duty, to secure hospital care and service, to make as much trouble as possible for those in authority, and to outwit them if possible. The chief causes are avoidance of danger and quest of a pension. . . . A successful malingerer promptly has a host of imitators."⁶⁹

Jones and Llewellyn mention the following motives of malingering: (a) monetary gain, (b) to evade punishment, (c) to evade duty or responsibility, (d) to evade or escape military service. And they note the following manifestations: pain, hyperesthesia, anesthesia, analgesia, limping gait, tremor, contractures, paralysis, epileptiform seizures, amaurosis or amblyopia, contractions of the visual field, deafness, aphonia, stuttering, mutism, deaf-mutism,⁷⁰ etc. Colonel Bailey mentions similar manifestations.⁷¹

In the case of legally established malingering which has come under my own observation the manifestations were typical of hysteria: shuffling, labored gait, stocking anesthesia, weakness, pain in legs and tremor. The case would, indeed, have passed for one of hysteria, had not the patient procured a fraudulent roentgen-ray plate. He had fastened a piece of bone to the sole of his foot with a strip of adhesive plaster which made it appear on the roentgen-ray plate as a detached piece of bone in the metatarsal region, an easily exposed deception. He was tried before General Court Martial, found guilty and given twenty years.

DISTINCTION BETWEEN HYSTERIA AND MALINGERING

The question thus arises, What is the distinction between malingering and hysteria? A search through the literature reveals but one point to which the differentiation is fastened almost unanimously, namely,

67. Jones, A. B., and Llewellyn, L. J.: *Malingering*, Philadelphia, P. Blakiston's Son & Co., 1918, pp. 117 and 118.

68. Bailey, Pearce: *Malingering in U. S. Troops*, Mil. Surgeon, March and April, 1918.

69. Ford, J. H.: *Details of Military Medical Administration*, Chapter on Malingering, Philadelphia, P. Blakiston's Son & Co., 1918, p. 531.

70. Jones, A. B., and Llewellyn, L. J.: *Malingering*, p. 12.

71. Bailey, Pearce: *Malingering in U. S. Troops*, Mil. Surgeon, March and April, 1918.

the *conscious or unconscious quality of the motivation*. Yet even on this point writers have shown much inconsistency; for it is generally admitted that even malingering may be "subconscious" or "unconscious."

The cardinal point of difference is that the malingerer simulates a disease or a symptom which he has not in order to deceive others. He does this consciously. . . . He lies and knows that he lies. The hysteric deceives himself by a mechanism of which he is unaware and which is beyond his power consciously to control.⁷²

It may require a rather exhaustive study to determine whether the symptoms are produced on the basis of a conscious or an unconscious wish, which is essentially the difference in etiology between malingering and hysteria.⁷³

All hysterical manifestations are of the nature of unconscious psychical reactions, just as voluntary acts are conscious psychical reactions.⁷⁴

Exaggeration, either conscious or unconscious, is the most common form of malingering.⁷⁵

Malingering, as well as lying and deceit, far from being a form of conduct deliberately and consciously selected by an individual for the purpose of gaining a certain known end, is in the great majority of instances wholly determined by unconscious motives, by instinctive biologic forces over which the individual has little or no control.⁷⁶

It is not always true that malingering is an acutely conscious reaction, as it is often beyond the awareness of the individual, occurring in the subconscious.⁷⁷

It is strange that so futile a consideration, one so obviously belonging to the domain of metaphysics and not science, as the question of degree of consciousness of a mental process, should become the preoccupation of scientific men and should be chosen as a criterion of clinical diagnosis!

Everyone who has given the matter any thought must be aware of the great difficulty of defining to oneself the motivations of even the simplest volitional processes. Such self analysis requires special skill and training in introspection; and even under the best conditions the data that thus become available can perhaps never become a part of the material of science, being serviceable only as *leads* to problems in psychology.

72. Salmon, T. W.: *The Care and Treatment of Mental Diseases and War Neuroses ("Shell Shock") in the British Army*, New York, The National Committee for Mental Hygiene, 1917.

73. MacCurdy, J. T.: *War Neuroses*, *Psychiat. Bull.*, July, 1917.

74. Jones, A. B., and Llewellyn, L. J.: *Malingering*, p. 117.

75. Jones, A. B., and Llewellyn, L. J.: *Malingering*, p. 59.

76. Glueck, B.: *The Malingerer; a Clinical Study*, *Internat. Clin.* **3**: S. 25, 1915.

77. Sheehan, R.: *Malingering in Mental Disease*, *U. S. Nav. M. Bull.* **10**: 646 (October) 1916.

One may, indeed, have an illusion of lucidity of consciousness; but he needs only to attempt a *formulation in language* of the results of his introspection to convince himself of the darkness within.

When we are dealing with weak-minded, ignorant, emotionally unstable, morally defective individuals, such as hysterics and malingerers; and when, moreover, the question is one of "conscious," "coconscious," "preconscious," "foreconscious," "subconscious," or "unconscious" *deception*, it is all the more amazing that a criterion, which is, in the first place, vague, and, in the second place, purely subjective, should seriously occupy the professional mind as a guide in practical work.

But there is more to add to the confusion. A case, it is said, may begin with conscious deception and end with unconscious self-deception; or vice versa; or there may be a mixture of unconscious and conscious simulation (i. e., hysteria complicated with malingering); or there may be a condition half-way between conscious and unconscious simulation.

M. Gilbert Ballet points out that a man may begin by shamming, and, as the result of auto-suggestion, end by developing the condition simulated, thus becoming the victim of his own chicanery.⁷⁸

Hysteria, when prolonged for months, is very apt to have malingering grafted on it, and what was at first a purely psychical disturbance of function may ultimately be perpetuated mainly by the intention to deceive.⁷⁹

One has to recognize the fact that a genuine hysterical person may for purposes of gain resort to simulation.⁸⁰

This condition (so-called guard house back or camptocormia), in which the back is held stiffly forward or laterally, is frequent in our army. . . . It is generally regarded as midway between malingering and hysteria.⁸¹

While it is in the nature of things that the motives actuating hysterical conduct should be, for the most part, concealed, this fact can by no means be regarded as proof of their being beyond the awareness of the patients. Occasionally they are either confessed or detected in other ways which show their clearly conscious nature, as in the following cases:

1. "To be honest about it, if I could get out of the army I would like it; now that's the truth. My half brother has a big farm, about seven or eight hundred acres of land and his wife is sick; and he has been at me to come and live with him, sort of look after his farming, his horses, and so on." (S., 7 Truck Co., 23 Eng. Case of hysteria characterized by pains in head and back, dizziness, amnesia, paralysis of legs, stammering, weakness, fatigability; cured by "the most earnest prayer.")

78. Campbell, H.: War Neuroses, Practitioner, London 96:501 (May) 1916.

79. Harris, W.: Nerve Injuries and Shock (Oxford War Primers), New York, Oxford University Press, 1915.

80. Jones, A. B., and Llewellyn, L. J.: Malingering, p. 129.

81. Bailey, Pearce: Malingering in U. S. Troops, Mil. Surgeon, March and April, 1918.

2. "I know I never worked hard in my life. If you'se want to discharge me I could work in an ammunition factory or something like that. I could go home then and be near my girl; I know I'd be more contented then. I might as well tell you the truth; no use of me lying." (D., 301 Co., Q. M. C. Case of hysteria characterized by limping gait, attacks of excitement.)

3. "About 6 o'clock on the night of April 12, on my way to the watch post—well, I can't say that I was blown up because I heard no explosion, and did not see the trench cave in, and had no blood drawn on me except a slight scratch on my back—I became unconscious, but I do not know the cause of my unconsciousness. When I came to I found myself between 30 and 40 feet in front of the trench in no-man's land. I managed to crawl back into the trench by using my hands and arms: I could not use my legs, as I had lost the use of both of them completely." The following version was later obtained by correspondence from First-Lieutenant Harold P. Gibson, 23 Inf., A. E. F.: "On April 12, 1918, while I with B. (the patient) and Corporal Hiller, both of my platoon, was inspecting outposts, B. suddenly stopped, fell against the side of a trench and told me he had no control over both his legs. He had to be carried to the battalion first-aid station and was then evacuated. B. had, during all the time that I knew him, been troubled with the arches of both feet." (B., Co. C, 23 Inf. Case of hysterical paraplegia, areas of anesthesia, violent tremors, globus, etc.)

Among other points of differentiation between hysteria and malinger which have been suggested are: 1. Result of treatment by persuasion, i. e., if persuasion fails to cure the case is not hysteria but malingering (Babinski). 2. A desire to be cured speaks for hysteria; the opposite indicates malingering. 3. The malingerer dreads examination; the hysteric welcomes it. 4. Hysterical manifestations bear the stamp of a certain genuineness which those of malingering lack. These points merit somewhat detailed discussion.

As regards result of treatment by persuasion, it is very generally recognized that in many cases which are by all diagnosed as typical hysteria persuasion fails to cure—so-called refractory hysterics. In other cases, also refractory, a cure is, indeed, obtained by persuasion but only when it is reinforced with painful electrical treatment (Kaufmann method, "torpillage"), ammonia, isolation and liquid diet, threat of operation, threat of court martial, etc.

It is true, of course, that many cases of hysteria have readily yielded to persuasion; but the conditions under which that has happened should be taken into account. When the danger was removed of being returned to the front, especially, as already stated, after the signing of the armistice, many cases were not only easily "persuaded," but were cured by any method that happened to be tried, though they had previously proved refractory. The cure in these cases is obviously to be attributed not so much to persuasion as to the removal of the danger. Some patients have remained refractory even since the signing of the armistice; in these cases the actuating motive is to gain government compensation; that is the reason why the "persuasion" that has cured thousands of others is doing them no good.

Moreover, proved malingering has also in many cases yielded to persuasion, while other cases have proved refractory; in other words, the experience with malingering, in that respect, has been exactly like the experience with hysteria. I would cite, in this connection, the observations of Sicard.⁸²

In six cases the deception was detected by the mild method (*méthode douce*). This consists in appealing to the pride of the patients, avoiding all suggestion of reproach, all criticism in public, and, above all, any accusation of malingering before a third party. We take them aside, and make them realize the unworthiness of their conduct. We appeal to their patriotism and prescribe an electric treatment to serve as pretext for a rapid cure. We have had, from some at least, expressions of real regret and remorse for their actions. These men were returned to the army. Two of them have written to us since and are giving good service at the front.

But other malingerers, those of fixation as a whole, remain inflexible. They exhibit an impervious obstinacy. One feels that all persuasion is useless, that every appeal to their better nature, to their moral sense, is vain. There is no worse deafness than that which is resolved not to hear. The only recourse is threat (*la manière forte*) of court-martial, or at least of appearance before court-martial.

The author describes in a note an interesting case of malingering in which a soldier terminated a period of ten months without a spoken word by a well-acted seizure in which speech and hearing supposedly returned to him. He confessed his trick later in a letter, but promised to atone for it by his future conduct—a promise which he is keeping.

Babinski's differentiating test of cure by persuasion is based on his general conception, according to which the essential feature of the hysterical personality is abnormal suggestibility. But a close scrutiny of the facts does not support this conception. An equally plausible case might be made out for abnormal *lack* of suggestibility.

Under certain conditions the hysteric is, indeed, remarkably receptive to *certain* suggestions; but he is at the same time refractory to others. When he has to play sick in order to avoid dangerous duty he will not only be readily influenced by suggestions unwittingly made by the examining physician in testing for disorders of sensation, etc., but will actually seek suggestion by observing cases of real disease and will develop by autosuggestion such symptoms as he may imagine to be appropriate for a "dead nerve," "stoppage of circulation," etc. At the same time he is apt to resist any suggestion of cure.

But a time comes when, on removal of danger, the situation changes. What was previously a life-saving disability now becomes a nuisance. Although many are, in the new situation, cured spontaneously, others feel that a sudden cure without treatment would but betray the false nature of the trouble. Nothing is more natural than that they should

82. Sicard, J. A.: *Simulateurs sourds-muets*, Paris méd., Oct. 23, 1915.

again seek the cooperation of our gullible profession to assist them in making a seemingly and plausible exit from an awkward situation. And so after taking electricity, hypnosis, reeducation, vocal exercises or what not, accompanied by "suggestion," they are pronounced cured; this carries with it not only relief from no longer serviceable disability, such as mutism, paralysis, contracture, etc., but also, by implication, added certification by duly constituted medical authority that a disease had existed.

Turning now to the next point of differentiation, according to which a desire to be cured speaks for hysteria, while the opposite indicates malingering, I have to say that no generalization in this respect can be made which would hold true for all cases of hysteria; everything depends on special features in a given case and on special circumstances.

Every practitioner knows the service a nervous illness often is to a patient in dealing with relatives, over whose head the patient holds it almost as a threat; this process may be consciously or unconsciously carried out. Under such circumstances the patient's deep-rooted objection to getting better may defy all therapeutic measures.⁸³

I have seen cases in which stubborn resistance to treatment gave way, following the signing of the armistice, to an impatient longing to get well. The following is a good example: "Oct. 7, 1918, electro- and hydro-therapeutic treatment discontinued because patient resists everything done for him and is sure nothing will help him. October 14, no improvement. October 24, no improvement. November 8, no improvement. November 18, transferred to Plattsburg."—(From the clinical record at Walter Reed General Hospital.) "This complete paralysis of the leg had continued for several months in spite of all efforts in one of the best French hospitals to effect a cure. He had been given some of their most drastic electrical treatment without obtaining any response. His desire to get well has been so great that after hearing another patient remark that he had been able to walk after falling down a flight of stairs, he tried a like cure on himself by throwing himself down a flight of stairs. In the first treatment, combined faradic-galvanic current, applied to the left foot, we were able to develop some voluntary motion. After the third treatment he was able to walk without crutches. In the course of about two more weeks (Jan. 3, 1919) he was discharged, cured."—(Report of Capt. Claude D. Kellam, M. C., chief of physiotherapy department, U. S. Army General Hospital No. 30, Plattsburg Barracks, N. Y. Case is that of S., Co. I, 103 Inf.)

The next alleged differentiating point, according to which the malingerer dreads examination, while the hysteric welcomes it, is, as far as my experience is concerned, untrustworthy. In the case of malingering cited above the patient at all times willingly submitted to neurologic examinations in which the areas of anesthesia were repeatedly mapped out, etc. Under similar conditions hysterics, too, welcome examination.

83. Jones, Ernest: *Papers on Psycho-Analysis*, New York, William Wood & Co., 1918, p. 354.

But I have many times seen hysterics cease to cooperate and become resistive to examination on a suspicion arising in their minds that the object of the examination is to test the genuineness of the symptoms. This was especially noted in cases of convulsions, in which the patients by turning away, biting, struggling, and fighting resisted an examination of the pupils, knee jerks, plantar reflexes, etc.

The last mentioned differentiating point, according to which hysterical manifestations bear the stamp of a certain genuineness which those of malingering lack, is also not to be relied on. All that can be said is that in both hysteria and malingering one meets with various degrees of adroitness in simulation, various degrees of determination and persistence.

Macdonald tells of a man, feigning epilepsy, who during a fit suffered without flinching knives thrust under his nails, the insufflation of irritating powders into his eyes, and one day fell 30 feet to convince the expert, though finally he acknowledged his deceit.⁸⁴

To sum up: my own experience, much discussion with other medical officers, and a study of the literature on the subject under consideration, all lead me to the conclusion that *what some have described under the name of hysteria and what others have described under the name of malingering are one and the same thing*. The difference seems to be entirely one of viewpoint.

"Hysteria" is an expression which would stress a medical viewpoint. "Malingering" is one which would stress a legal viewpoint.

SEX FACTORS—INTRAPSYCHIC CONFLICTS

There remain two other point in connection with hysteria which merit discussion in the light of the war experiences: (1) the part played by sex, (2) the theory of intrapsychic conflicts.

Although Freud's views as to the exact part played by sex factors in hysteria have undergone considerable modification from the time of his original formulation nearly twenty-five years ago, even in his recent formulations the sex element is regarded as essential in the etiologic mechanism. The hysterical symptom corresponds to the return to a manner of sexual gratification which was real in infantile life and which has since been repressed. The hysterical symptom can assume the representation of various unconscious non-sexual impulses but cannot dispense with a sexual significance.⁸⁵

It seems quite probable that, in relation to a certain variety of material, the idea of the universality of sex factors is well founded. The sphere of sex, under ordinary conditions, might even *a priori* be

84. Jones, A. B., and Llewellyn, L. J.: Malingering, p. 48.

85. Freud, S.: Sammlung kleiner Schriften zur Neurosenlehre, 2nd Ser., 1909, pp. 142, 143, 150; cited by Pfister, O.: The Psychoanalytic Method, New York, Moffart Yard & Co., 1917; transl. by C. R. Payne.

regarded as the main if not the sole source of "concealed, illicit, morally untenable motives" postulated by me as the mainspring of hysterical conduct. But the war experience has shown even to loyal adherents of Freud that hysterical manifestations can be actuated by motives other than sexual.

Psychoanalysts in civilian practice claim that the individualistic tendencies in question are preponderantly related to the sex instinct. In war, however, this does not seem to be the case, these latter tendencies coming into play, apparently, only as a complication.⁸⁶

The psychoneuroses of peace time are usually the result of mental conflicts between repressed instinctive tendencies and the demands of civilized life. The instinct most often involved is the sexual. In the psychoneuroses of war time, however, we find that the conflict is usually between the soldier's ego instincts (particularly that of self-preservation) and the demands of military service.⁸⁷

Even in peace times, neurologists have seen but too often hysterical manifestations ("traumatic neuroses") arise on the basis of exaggerated claims for indemnity, sick benefit, accident insurance, workmen's compensation, etc., without the intervention of sex motives.

It seems, therefore, justifiable to conclude that an illicit motive is an essential part of the mental mechanism of hysteria; but such motive need not be of a sexual nature, although undoubtedly it very often is.

In most other respects, what I have seen of hysteria in the course of my war experience bears out Freud's conception of its underlying mechanisms more fully than any other pre-war conception. I would cite here the well formulated presentation by Ernest Jones:

Neuroses are not diseases or accidents that happen to a person, as the French school of psychopathology maintains, but are phenomena produced and brought about by some tendency in the person's mind, and for specific purposes. Freud distinguishes three classes of motives that operate in this way, one essential, the other two not. The indispensable one is an unconscious desire to obtain pleasure by gratifying in the imagination some repressed and disassociated impulse, a motive, therefore, arising in the part of the mind that is not in harmony with the ego-ideal. A second motive is to achieve some end in the outer world; for instance, sympathy from an unkind husband, which the person finds easier to do by means of a neurosis than in other ways. The third set of motives has the same purpose as the last, but may be distinguished from it in that they concern the making use of an already existing neurosis rather than the helping to bring one about.⁸⁸

Turning now to the subject of intrapsychic conflicts, it will be remembered that the manifestations of hysteria are regarded by some as a

86. MacCurdy, J. T.: War Neuroses, *Psychiat. Bull.*, July, 1917.

87. Payne, C. R.: Special Report on the Applications of Psychoanalysis to War Neuroses at U. S. Army General Hospital No. 30, Plattsburg Barracks, N. Y.

88. Jones, E.: *Papers on Psycho-Analysis*, p. 573.

sort of compromise resulting from a conflict between repressed, sub-conscious wishes and the patient's conscious tendencies representing the better part of his "split-up personality."

Flight or desertion is rendered impossible by ideals of duty, patriotism, and honor, by the reactions acquired by training or imposed by discipline and by herd reactions. Malingering is a military crime and is not at the disposal of those governed by higher ethical conceptions. Nevertheless, the conflict between a simple and direct expression in flight of the instinct of self-preservation and such factors demands some sort of compromise."

The manifestation of abnormally repressed mental processes is to be understood only by consideration of the action of intrapsychical conflict. . . . The energy finds an outlet in some somatic manifestation, a process Freud terms "conversion." This is the characteristic mechanism underlying hysterical troubles, where a given bodily symptom, such as a tremor or an aphonia, is the expression of a repressed mental complex."

I can confirm, from such observations as I have been able to make, the existence of a conflict. But it has seemed to me to be, for the most part if not entirely, a conflict rather between the patients' desire to shirk, loaf, avoid exposure to danger, gain unearned compensation, etc., and pressure from external sources the object of which might be to expose their motives and the unreal nature of their disability, to bring on them the opprobrium of their comrades, to render them liable to legal prosecution, etc.

In other words, I was unable, in the great majority of cases, to detect any pricking of conscience, evidence of regret at being a burden rather than a help to their country in its great emergency, any struggle between nobler and baser parts of self, but rather a general lack of evidence of the existence of a nobler self in these cases.

This brings us to the subject of the hysterical personality.

THE HYSTERICAL PERSONALITY

I have already endeavored to show that hysteria is but one of a number of varieties contained in the large group of neuropathic constitutions. The family and personal histories of hysterics indicate some sort of relationship to the constitutional psychoses, epilepsy, mental deficiency, constitutional psychopathic states, etc. But the hysterical personality can be more specifically defined. Its essential feature, it seems to me, consists in a *character defect*. In the moral side of our nature three motivating principles can be distinguished, each of which actuates our conduct in a measure which differs in different individuals.

89. Salmon, T. W.: The Care and Treatment of Mental Diseases and War Neuroses ("Shell Shock") in the British Army, New York, The National Committee for Mental Hygiene, 1917, p. 30.

90. Jones, E.: Papers on Psycho-Analysis, p. 23.

The first of these may be termed pure or esthetic morality; it is represented in the saying "It is better to be right than to be president." No considerations of selfish advantage, of mere catering to popular taste or demand, are here permitted to enter. A person actuated by this principle turns away from thoughts of deception, theft, dishonesty, or any other moral filth, just as he might, from inherent esthetic repulsion, turn away from a foul smell.

The second principle may be termed prudent morality; it is represented in the saying "Honesty is the best policy." Unlike the case of the first principle, here considerations of selfishness and personal ambition not only are permitted to enter, but are the basis of doctrine. A person actuated by this principle turns away from wrongdoing not from an esthetic aversion, but because of a conviction that, in the long run at least, it does not pay.

The third principle, imposed morality, has its roots in the deterrent force of such measures of redress, retaliation, or protection as are available to individuals and society in dealings with wrongdoers. A person actuated by this principle has no esthetic aversion to wrongdoing; and he regards the maxim of prudence with cynicism. His preoccupation is, mainly, how to escape detection, conviction and punishment. If he refrains from wrongdoing, it is only when the risk involved is too great and too immediate.

I could not better define the hysterical personality, as I have observed it, than by saying that it is characterized by total lack of the first principle; that it is at best actuated by the second principle; and that it is, in its typical manifestations, actuated entirely by the third principle; i. e., in so far as its conduct has any moral quality at all.

This places the hysterical individual in close relation to the criminal. Yet a certain difference may be pointed out. Most hysterics are characterized by a trait which is foreign to many criminals: indolence.

A desire to lead a parasitic existence, to be a burden on relatives, employers, the government, to live on a pension and do no work, is characteristic of many of these patients. They would, and often do, steal anything conveniently within reach, lie, cheat, make work and trouble for others, wantonly destroy government property, but they haven't the enterprise or energy that some criminals have of planning and carrying out an embezzlement or a burglary or a train robbery: That is too much like work.

This description may seem to some much overdrawn. I would, therefore, again note that the above described traits of hysterical personality exist in all degrees. Between the man of highest integrity, actuated only by the purest motives of unselfish service, and the one who utterly lacks all moral compunction and is constantly preoccupied

with motives of shirking and of organizing a parasitic existence, there are many shades of transition.

It should, moreover, be borne in mind that the material observed in the Plattsburg Hospital, on which this study is based, represents, by selection, the most refractory cases of hysteria met with in the army. The reader will recall that of 100 so-called concussion and nervous cases received at triages near the front, sixty-five were returned to duty within a few days and thirty-five were sent to army neurological hospitals. Of the latter, twenty were later also returned to duty and fifteen sent to Base Hospital No. 117, at La Fauche, in the Service of Supplies. Eventually fourteen of even this group were returned to duty and but one sent back to the United States.⁹¹

My comments refer particularly (a) to this 1 per cent. of cases, weeded out from amongst all concussion and nervous cases on the basis of their stubborn resistance to treatment and total unfitness for duty and, (b) to a small number of still more flagrant cases that had arisen in the domestic cantonments. To what extent generalization from my conclusions might be valid is, of course, another question.

The statistical analysis, in the beginning of this section, of two selected groups of cases of hysteria, demonstrates differences in degree. Analyses of individual cases present such differences even more strikingly.

One man, for instance, gives up a well paying position and enlists voluntarily in the Signal Corps with the hope of "helping in the war and at the same time maybe learning something," though determined to avoid, if possible, combatant duty or exposure to any danger. Following an unexpected order to prepare for embarkation for overseas he develops violent coarse tremors and fainting attacks which persist until his transfer is arranged to the medical department with assignment to duty in the Plattsburg hospital. Thereupon, being assured of the removal of the danger of exposure to submarine attack, etc., he makes a prompt and complete recovery, and renders daily extremely valuable and conscientious service as office assistant. (R., Co. G, 321 F. S. Bn.)

In contrast with this case may be cited that of a man who became inspired with the idea of utilizing this rare opportunity of securing a life pension. With that in mind he enlists and two weeks afterward develops, from an alleged injury due to being struck in the groin by a case which he was carrying, a stooped over position, so-called camptocormia. Shortly afterward he is operated on for varicocele, and this aggravates his disability. Thereafter he spends over fifteen months, i. e., the remainder of his "service" in the army, in hospitals or on furloughs for his health, does no work, makes all kinds of trouble for the authorities, declares he can never be cured, demands discharge on certificate of disability: "If the government is afraid of a little compensation, I don't think it is treating the soldier boys very fair." (S., 4th Co., B. S., S. C.)

91. Editorial, *Mental Hygiene*, January, 1919.

NEW DESIGNATION SUGGESTED FOR HYSTERIA

From all that has been said above, the reader can almost surmise the view to which the war experience has led me. I feel, on the one hand, that the practice of camouflaging the true nature of the conditions here dealt with by means of such euphemisms as hysteria, war neurosis, concussion neurosis, traumatic neurosis, shell shock, etc., and thus, through implied authoritative professional support, imparting to them a stamp of respectability, is bad in every way. On the other hand, in view of the fact that, whether conscious or unconscious, simulation of disease occurs on a basis of inborn neuropathic constitution, it cannot be encompassed within the simple formula of malingering, i. e., crime, the responsibility for which is entirely on the patient. I would banish from medical classification the above named terms and others like them and designate the cases in question *constitutional psychopathic state, simulation*.

NEURASTHENIA

The cases classed under the heading of neurasthenia may be roughly divided into two groups which are superficially very similar to each other but which, on closer study, may be found to present wholly different psychic mechanisms.

The first of these is characterized by depression, discouragement, difficulty of concentration, feeling of inadequacy, and psychomotor retardation—in other words, it is a condition apparently allied to the manic-depressive psychoses.

The second is characterized by vague general hypochondriasis with purely subjective symptoms, and may often be shown to be motivated exactly in the manner of the above discussed hysterical manifestations. It is, to my mind, but a special type of hysteria or simulated disease. It is apt to be seen in persons presenting the same sort of character defect as that which underlies common hysteria, but endowed with better intelligence, of better education, more polished and diplomatic, more suitable and plausible. Thus I find it relatively more frequent in commissioned officers than in enlisted men.

My experience has amply shown that the possession of native intelligence far above the average and good educational and social opportunities are not incompatible with gross lack in moral sentiment. The following cases are cited for illustration.

1. An officer who had been under suspicion and surveillance was later reported in France for what seemed to be pro-German propaganda. He had stated that enormous supplies had been abandoned by the Allies to the Germans in the spring of 1918; that the French troops had become utterly demoralized; that he had been directed to place time bombs for the destruction of the supplies of the Allies in the event of further advance by the Germans; that the fall of Paris was only a matter of days; that the Germans had a tremen-

dously powerful new explosive, etc. He was placed under arrest on June 5, 1918; later, following examination by a psychiatrist, he was declared not fully responsible and returned to the United States as a nervous patient (K., A. S. S. C.).

2. An officer who gave a history of fainting spells and other epileptiform manifestations which, he said, began after he had been gassed in France, showed nothing on examination. Aside from his history there was no evidence of epilepsy and no record of his having been observed in seizures of any kind. Shortly following his admission to this hospital, papers were received showing that he had been charged with a serious offense in France and had disappeared for a time. His whereabouts were for a time unknown, but he appeared in Field Hospital No. 6, later placed in Class "D" by medical officers and returned to the United States (R., 4 Eng.).

STRIKING CASE DEMONSTRATING THE REMARKABLE STABILITY
OF A NORMAL NEUROPSYCHIC CONSTITUTION

On Nov. 18, 1918, a group of men, survivors of the torpedoed transport *Ticonderoga*, were admitted to the Plattsburg Hospital. It would seem that nothing that happened in the war was more exhausting, horrifying, depressing, and nerve racking than the experience of the men on the *Ticonderoga*. The abridged account furnished by one of these men is herewith submitted. This case is instructive in that it shows what remarkable stability a normal mental constitution has and how inadequate etiologically, in the absence of a neuropathic predisposition, are the factors to which so often psychoses and neuroses are attributed.

H., white, aged 18; previous history negative; enlisted July 15, 1918, and sailed for overseas Sept. 18, 1918. The transport was shelled, torpedoed, and sunk on Sept. 30.

At about 5 o'clock in the morning, patient was in his bunk with all his clothes on but his blouse and shoes; he was suddenly awakened by the bell signal "to life boats" and by the noise of constant shelling. He slipped on his blouse, shoes and life preserver; as he reached the deck he saw one shell knock the 3-inch gun off the platform and kill the gun crew; he was knocked down by the jar though not hurt or rendered unconscious; started toward the upper deck, the shelling going on all the time; one shell exploded about 10 or 15 feet from him; this time he was rendered unconscious and, on coming to about five minutes later, found himself in a narrow passageway into which he had been thrown. He had a violent headache, was gasping for breath, shaking violently all over, and found that his hands, arms and body had been sprayed with minute particles from the exploding shell, which were so hot that his clothing was charred or burned through where they struck. For about an hour he assisted in preparing the life-boat for lowering, loading it with blankets, etc., the shelling going on all the time. The submarine was seen about 200 yards away; the bridge had been partly blown down; shells were seen striking various parts of the ship and throwing up and killing men; some life boats which had been lowered were struck by shells and sunk; others were dumped on account of being lowered in a hurry; wounded men were all over the deck, and many, both wounded and not wounded, were in the water;

One man had had his nose shot off and received a gash in the back of his head; he approached the patient and asked that he kill him. There was a good deal of shrieking and groaning, although most noises were drowned in the noise of constantly exploding shells. Then a big blaze was seen over what was left of the bridge and all men were ordered to fight the fire on the bridge. The shelling stopped for about twenty minutes but started again from the other side. While lowering their life boat a shell killed the paymaster, a sergeant, a private, and some others who were standing there waiting to get in. No other life boat succeeded in getting away and only one raft. In the meantime the *Ticonderoga* was struck by two torpedoes and finally sank, carrying with it the wounded on board.

When they began rowing away with their life-boat there were twenty men in it. In a few minutes they saw the submarine coming toward them. The captain (Capt. M., ranking as Lieut.-Com. in U. S. Navy) said, "Well, we shall die like men!" (He had been severely wounded in both legs, with face bleeding, and part of the time unconscious; later at times delirious.) At the same time they rowed toward the submarine and soon came alongside of it. One of the submarine officers asked them first, in broken English, if they were armed; one ensign and one soldier were then taken on board the submarine; in the meantime the life-boat was tied by a tow rope to the stern of the submarine; then the submarine submerged, but fortunately the knot became untied and the life-boat was left afloat with eighteen men in it, of whom five had been wounded. They floated around all day; at about 3 o'clock in the afternoon sighted a raft, and toward 5 o'clock came near enough to it to throw a rope to the men on it; there were ten men on the raft, some of them wounded; they had no water or rations. Five of the men from the raft succeeded in getting into the life-boat; it was not considered safe to permit any more to come in. They planned then to tow the raft so as to have the men near enough to give them rations, but the raft slipped away, and they lost sight of it. One of the wounded men in the life-boat died that evening and his body was thrown overboard. The first two days and nights it was rather rough and it rained; it kept two men busy all the time baling. The canvas covering the boat had to be held down at the gunwale to prevent being swamped by a wave; the sail that had been put up had to be held by a rope and managed as was necessary. Two of the severely wounded men lay in the bottom of the boat and got a little sleep; but the remaining men got no sleep whatever in the four days and three nights.

During the first night the patient and several of the others kept vomiting small quantities of greenish liquid. Nobody seemed hungry, and they ate nothing until the afternoon of the second day, about 3 o'clock, when they had one apricot, one small hard-tack, and some apricot juice mixed with water—only a tablespoonful of that mixture to each man. Everybody was thirsty, but only small quantities of water were given out, and those only at meal times. The third and fourth days they had one meal each, same as above and, in addition, one-fourth of a thin slice of bologna. All the time they were shaking with chilliness and were sopping wet. The fourth day, however, was much calmer, sunny and warm, and they got dried out and warmed up. At noon on the fourth day they sighted a ship (*The Moorish Prince*) in the distance and at 3 o'clock they were taken on board.

The patient suffered from headache, great weakness, shook all over, and had pain in the back from cramped position; he was also famished and extremely thirsty. The others were in the same condition. They got some

whisky, were cautiously fed and were placed in warm bunks. The patient's sleep was disturbed by dreams of shelling, etc., and he would start suddenly; also every noise, especially the boat whistle, would wake him with a start; although he was able to be up and around, he lay in his bunk the greater part of the time.

On Sunday, October 6, their fourth day on the freighter, H., with others, was transferred at sea to a British transport and reached New York on October 10. He was taken to the U. S. Army Debarkation Hospital at Ellis Island, New York City; after two nights there the patient was transferred to Camp Merritt, N. J. He complained of headaches daily, nervous shakiness, bad dreams and being readily startled by noises. On November 17, he was transferred to the Plattsburg hospital.

Physical Examination on Admission.—His height was 67½ inches, his weight 139 pounds in ordinary clothing. (His usual weight is 150 pounds.) The patient stated that he tires easily. He had rather pronounced tremor of hands and eyelids. (Two days later the tremor was greatly reduced.) The grips were equal but rather weak. Findings otherwise were negative.

Mental Examination.—"I feel quite well now; I still have headaches, but not very often. At times I have a feeling of longing and worry due to the fact of being away from home and facing death as I did and not having seen my people. The dreams that I have now are only like those I used to have before. I am not fit for full duty, but could do light duty and I think I would be able to work up to full duty before long." His attitude and manner were entirely normal. The patient asked for his clothes and to be given something to do. He adapted himself well to the hospital routine; presented no emotional disturbance, disorder of flow of thought, oddities, hallucinations or delusions. Orientation, memory, grasp on general information were intact. He was unable to recall any dream.

PSYCHO-ANALYSIS IN WAR NEUROSES

The Plattsburg hospital has been fortunate in having on its medical staff an able exponent of psycho-analysis, Dr. Charles R. Payne. The report submitted by him contains conclusions which are worth noting:

Psychoneuroses of war differ from those of peace in: (1) the conflict involves the ego instincts rather than the sexual; (2) in the war psychoneuroses the conflict is much nearer the surface of consciousness. Psychoanalysis is not practicable in treating cases of war psychoneuroses among private soldiers for three reasons: (1) too much time required; (2) patients during war lack real desire to get well; (3) majority of patients are not of sufficiently high intelligence to be suitable for psychoanalysis. Psychoanalysis is of great value in treating psychoneuroses among officer patients.

LESSONS FOR CIVILIAN PRACTICE

War neuroses and malingering in the army have their counterparts in civilian life—hysteria and the so-called traumatic neuroses. The problem of "traumatic neuroses" is very likely to grow to vast proportions with the progress of legislation for workmen's compensation, accident insurance, sick benefit funds, etc. It would seem that the interests of society, insurance organizations, public service corporations,

etc., should be protected by amending legislation so that, while providing fully for compensation in cases of actual injury or disease, it should render the subject of purely functional, mental, simulated disability or disease entitled to no indemnity but only to examination and treatment in a hospital for a limited period of time.⁹² The removal of temptation would result in greatly lessening the incidence of simulated disability or disease.

SUMMARY

1. Hysteria is purely functional in its nature and the mechanisms underlying its manifestations are entirely mental.

2. By way of preliminary to the discussion of my own experience a brief restatement is offered of pre-war current conceptions of hysteria.

3. An analysis of two selected groups of cases of hysteria is offered—fifty cases in each group. This analysis leads to the following conclusions: (a) Hysterical phenomena are a special group of manifestations arising on a basis of neuropathic constitution. (b) Neuropathic heredity is probably the essential factor in the etiology. (c) The frequent occurrence of dissimilar heredity and, in the patients themselves, or neuropathic traits not specifically hysterical, points to a relationship of some sort between hysteria and other manifestations of neuropathic constitution. (d) The hysterical tendency varies widely in degree and shades off toward normal constitution by gradual transition.

4. Causes to which patients attribute their hysterical symptoms are obviously and absurdly inadequate; they are, at most, occasions out of which have arisen suggestions in part determining the particular symptomatology.

5. Various causes assigned by medical officers are enumerated. With the progress of the war, the view gained ground that physical factors, as such, played no part in the etiology of hysteria.

6. In studying the etiology of war neuroses, it is important to distinguish the acute emotional disorders observed at the front from hysteria.

⁹². Recently the head of the medical department of the French army requested the advice of the Société de Neurologie de Paris concerning the proper medicomilitary disposition of cases of hysteria still under military jurisdiction. The answer was threefold, the third part being: "In general, purely hysterical manifestations entitle one to no recompense. Exceptionally, a percentage of disability may be allowed somewhere between zero and 20 per cent., but in no cases shall the latter figure be exceeded" (Rev. neurol., 1919, No. 3). From this it would appear that the opinion of the members of this learned society does not much differ from mine.

7. According to Léri, it is a mistake to think that hysterical manifestations are an integral and necessary part of the emotional syndrome; they can appear independently of all emotion; and the emotional syndrome has nothing in common with hysteria.

8. Psychic factors to which war neuroses in general have been attributed—fright caused by danger from projectiles, horrifying sights, etc.—play a part only in the acute emotional syndrome; hysterical phenomena are not directly produced by them.

9. Those disorders which developed at the front and are the direct expression of violent emotion are never of long duration; the intensity of their manifestations subsides rapidly and almost all cases can be returned to the front within a few days.

10. Speaking with special reference to my experience, the main-spring of hysterical conduct consists in a concealed, illicit, morally untenable motive.

11. Its most frequent variations are: (*a*) To evade the law of conscription, (*b*) to procure, on reporting for physical examination at a training camp, rejection for physical unfitness, (*c*) to evade dangerous, disagreeable, or difficult duty, or to evade all duty, (*d*) to procure the ease and privileges of hospital care, (*e*) to procure discharge on certificate of disability, (*f*) to procure compensation for disability.

12. That illicit motive and it alone is the factor which actuates hysterical conduct, is further shown by the following three groups of facts: (*a*) Many cases have arisen in National Army cantonments in this country very early in the course of training. (*b*) Among prisoners of war who have been, like other soldiers, exposed to shell fire, strain, etc., scarcely any cases of hysteria or other psychoneuroses have been observed. (*c*) Very often quick and complete recovery from hysterical symptoms, having occurred on evacuation to a base hospital, is followed by a return of the symptoms in the same or even a greater degree of intensity, or by development of new symptoms, on any prospect arising of being sent again to duty.

13. Statements which have been made to the effect that war neuroses had not been observed in previous wars in such large numbers are probably not in accord with facts.

14. The factors to which patients themselves attribute cures are apt to be, like the causes they assign for their troubles, trivial and inadequate.

15. Among medical officers there is a striking general tendency to ignore mental mechanisms; cures are attributed to this or that therapeutic method, every method having its advocates and opponents.

16. According to my experience, the particular method of therapy is a matter of comparatively little importance.

17. In cures the following factors are frequently seen to be operative: (a) Medical officers impressing patients in such a way as to preclude any hope of successful imposition, (b) demonstration of the unreal nature of the disability, (c) strict discipline as opposed to sympathy, coddling, or humoring, (d) painful or otherwise disagreeable treatment, (e) removal of motive by change of situation.

18. Many spontaneous cures occurred, in previously refractory cases en route to the United States and later on the signing of the armistice.

19. Among circumstances contributing to the prevalence of war hysteria is gullibility of the medical profession.

20. As opposed to laymen, the tendency of medical men is to overlook or ignore underlying motives and mental mechanisms and to allow themselves to be imposed on in the crudest fashion.

21. Military law places medical officers in a difficult position. They must either designate the disability by some respectable name, such as hysteria or psychoneurosis, or make a diagnosis of malingering and have to prove criminal intent.

22. All are agreed as to there being a close similarity in the clinical manifestations of hysteria and malingering and as to there being great difficulty in practice of establishing the differentiation.

23. The motives which I have found to be the mainspring of hysterical conduct are the same as those which students of malingering have reported as actuating their subjects.

24. In the one case of malingering which has come under my own observation the manifestations were typical of hysteria.

25. A search through the literature reveals but one point to which the differentiation is generally fastened; namely, conscious or unconscious quality of the motivation.

26. It is strange that so futile a consideration, one so obviously belonging to the domain of metaphysics and not science, as the question of degree of consciousness of a mental process, should become the preoccupation of scientific men and should be chosen as a criterion of clinical diagnosis!

27. Further phases of confusion of hysteria and malingering.

28. Among other suggested points of differentiation between hysteria and malingering are: (a) Result of treatment by persuasion, i. e., if persuasion fails to cure the case is not hysteria but malingering (Babinski). (b) A desire to be cured speaks for hysteria; the opposite indicates malingering. (c) The malingeringer dreads examination; the hysteric welcomes it. (d) Hysterical manifestations bear the stamp of a certain genuineness which those of malingering lack.

29. In my experience differentiation based on any of these points is not possible.

30. My own experience and study lead me to the conclusion that what some have described under the name of hysteria and what others have described under the name of malingering are one and the same thing. The difference seems to be entirely one of point of view.

31. War experience has shown that hysterical manifestations can be actuated by motives other than sexual.

32. In most other respects, what I have seen of hysteria in the course of war experience bears out Freud's conception of its underlying mental mechanisms better than any other pre-war conception.

33. The essential feature of the hysterical personality seems to consist in a character defect.

34. I would banish from medical classification such euphemisms as "hysteria," "shell shock," "traumatic neurosis," etc.; and would also banish the expression "malingering" with its implication of crime for which the responsibility is entirely on the patient.

35. I would designate, instead, the cases in question by the term *constitutional psychopathic state, simulation*.

36. The cases classed under the heading of neurasthenia may be roughly divided into two groups. The first represents a condition allied to the manic-depressive psychoses; the second is characterized by vague general hypochondriasis, may often be shown to be motivated exactly in the manner of ordinary hysterical manifestations, and is, to my mind, but a special type of hysteria or simulation.

37. My experience has amply shown that the possession of native intelligence far above the average and good educational and social opportunities are not incompatible with hysterical character defect and with gross lack of moral sentiment.

38. Judging from the narrative of survivors of a torpedoed transport it would seem that nothing that happened in the war was more exhausting, horrifying, depressing and nerve racking than their experience.

39. These cases show what remarkable stability a normal neuro-psychic constitution has and how inadequate etiologically, in the absence of a neuropathic predisposition, are the factors to which psychoses and psychoneuroses are so often attributed.

Acknowledgement is cheerfully made of the great assistance derived from *Neuropsychiatry and the War*, a Bibliography with Abstracts, prepared by Mabel W. Brown and Frankwood E. Williams, and published by the National Committee for Mental Hygiene, New York.

Abstracts from Current Literature

THE ROLE OF FOCAL INFECTIONS IN THE PSYCHOSES. HENRY A. COTTON, *J. Nerv. & Ment. Dis.* 49:177 (March) 1919.

Cotton deserves credit for following the bent of the times and advocating that psychiatrists, too, pay attention to focal infections in cases of insanity, just as one would pay attention to more overt medical situations. No one will take issue with him on this. But he goes much farther than that. One has the feeling all through the paper that to Cotton focal infections are the real cause of his psychoses. To be sure, he says at one point, merely that they play an *important* rôle in the etiology of psychoses, and again that he is willing to give psychogenic factors their etiologic rôle when they exist, but that he does not believe they are essential for the production in all cases. He believes, however, that mental causes do not act in a psychogenic manner but as in tuberculosis, as he says, by merely weakening the patient's resistance toward physical factors. So far, then, he is fairly conservative. On the other hand, however, he also makes the statement that he is "inclined to place heredity and constitution in a minor rôle as determining the individual's reaction to certain toxins, but not determining the psychoses without these extrinsic factors." These are therefore practically flatfooted statements that mental causes do not act psychogenically and that focal infections, or to be quite just to Cotton, "extrinsic" factors, are the real causes in manic-depressive insanity. The reviewer is personally impressed with the importance of psychogenic causes per se, knowing of course full well that they are often difficult to find, especially by a superficial examination, and he would of course not deny the importance to physical factors, because we all have seen manic-depressive attacks following acute diseases. But to Cotton, evidently these are the real causes. One is, of course, open to conviction and therefore it is interesting to see what these claims are based on. He has eleven patients; five of these died. Case 1 was that of a woman who began with delirium after acute articular rheumatism. The delirium was protracted and left her apathetic (hardly a clear manic-depressive case). Finally she died with a streptococcus meningitis. Case 2, after "grip," became, probably, manic. The throat was found inflamed and viridans was discovered in cultures from the throat and the blood was positive for the same organism. She died twelve days later from pneumonia in which viridans was found at the necropsy. Case 3, four weeks after "grip" and pleurisy, became manic. "Physical condition suggested paresis." Bad teeth and cheek infection with streptococcus longus in cultures from cheek, mouth and blood stream were found. After nine days death occurred from endocarditis and general septicemia. Case 4, after ischiorectal abscess, presented a "typical manic reaction," to which delirious features later were added. He died in five days with pus in chest muscles inguinal region, purulent pleuritis, pneumonia, catarrhal enteritis, staphylococcus aureus being found in all abscesses. Case 5 was that of a nurse of peculiar make-up whose sister had trouble about some accusation she made against a man. After some days the sister was found with her wrist cut and the patient "as a raving maniac." On the second day of admission she developed a high temperature, pulse 140, convulsions, was semistuporous, in a "manic delirium," and soon developed jaundice and died. She had sordes and inflamed throat; the fixation test for streptococcus was positive. At the necropsy the

Connellan-King gram-negative diplococcus (designated by Cotton C-K diplococcus) was found extensively. The diagnosis of manic-depressive insanity may fairly be questioned, and as regards all of these cases it must be admitted that for the theory of manic-depressive insanity in general they prove nothing.

Case 6 was that of a man who in the previous year had had a depression. The attack under question was evidently a hypomanic state. During the attack teeth infection was discovered with the Connellan-King organism which was also found in the urine. After the extraction of the teeth he calmed down and developed a mild depression. Case 7 was that of a man who had had repeated exhilarations followed by another hypomanic attack in which a "highly acid stool" with Connellan-King diplococcus was found. Alkaline rectal injections and rest were prescribed for him. Under the treatment the hypomanic symptoms disappeared; but here again the condition was soon followed by a depression. It is, of course, not clear why, if these manic attacks are regarded as due to infection, the depression should occur after the infection is treated. Moreover, Cotton's statement that since these two cases were so similar one should expect similar infection (as was actually the case) again shows how close he regards the relationship between infection and the clinical picture—evidently to the extent of intensity and symptom picture. It must be added that Cotton himself seems a little doubtful regarding the convincing nature of these two cases, as he also seems to be about the next, that of a woman who developed a manic attack after pneumonia, and two weeks after a lumbar puncture recovered. Indeed he seems to have the same misgivings about Case 9, a woman who during a depression made a suicidal attempt (throwing herself from a bridge), after which she was confused and agitated. Since she showed evidence of intestinal intoxication and a streptococcus of the viridans variety was isolated from the stools "in much larger quantity than normally," she was given colonic irrigations. After this was done "for some time" she recovered, five months after admission. Curiously enough he seems to feel no doubt about the next patient (Case 10), a man with a long depression in whom fixation for viridans was positive in blood and fluid and whose teeth were infected. Although the latter were extracted, he did not improve. Vaccine from viridans was given, but he did not recover from the depression until months later. Case 11 is that of a woman who had had repeated depressions and one manic attack. With her psychosis were usually associated headaches and gastro-intestinal symptoms. In another hospital she was manic, depressed, stuporous and again delirious. Under observation she seems to have had a prolonged stupor. After six years she gradually recovered after extraction of one infected tooth.

The reviewer has purposely refrained from expressing an opinion about the bacteriologic side of the paper, as he feels hardly competent to do so. He thinks the cases have been fairly summarized. For Cotton's therapeutic endeavors they speak well, and as such are worth reporting, but they certainly do not justify the rather sweeping attitude which he assumes in regard to the theory of manic-depressive insanity in general.

Hoch, Montecito, Calif.

APHASIA AND ASSOCIATED SPEECH PROBLEMS. MICHAEL OSNATO, M.D., *Neurol. Bull.* 1:407, 1918; 2:19, 1919.

In this somewhat extended article the author endeavors to cover the whole field of his subject in rather an informal manner. He admits that he has little that is new to offer and he confines himself to a thorough review of

the literature interspersed with reports of cases of his own to prove or disprove the contention of the various authors. His arguments are based mainly on the didactic clinical grounds, for in but few cases has he pathologic evidence of the causative lesion.

The article starts with a complete and interesting review of Villiger's conception of speech development, but he doubts the truth of Villiger's contention that the development of intelligence and of speech are entirely independent. In fact, the author's main contention is that the two are inseparable.

Wernicke's idea of aphasia in its various types are reviewed, with little sympathy, and the author closes his first chapter with a reference to the speech defects other than aphasia, i. e., stuttering, stammering, agramatism, hearing mutism, congenital word blindness and deaf mutism.

In the second chapter the author proceeds with the development of his own ideas and those of others. He shows much sympathy with Head's review of Jackson's work and Marie's conception, except that of the much debated "lenticular region." He lays considerable stress on Burckhardt's operations of removal of speech center which resulted in no aphasia and in this connection he reports his first case—one in which a large part of the left temporal lobe sloughed away, following an exploratory operation. After one week this patient showed some paraphasia which the author considers to have been due to pain, fatigue and exhaustion rather than to the organic brain defect. Three months later the patient showed no aphasia. In reporting this case, the author outlines his method of studying his material, which consists of the fifteen questions proposed by Tilney.

The author now considers the question of apraxia, and quotes the opinions of Marie, Von Monakow and Grasset as evidence for his belief that both apraxia and aphasia are defects of the intelligence. In this contention he reports three cases with aphasia and intelligence defects with and without paralysis. In a fourth case aphasia and apraxia both existed with intelligence defects. There appear to be little grounds for believing in a local lesion as the cause of apraxia and the author maintains that "the whole cortex is indivisibly associated with the development of the function of specialized purposeful movement and its performance." In reviewing Kennedy's ideas of "stock brainedness" the author takes exception to his conclusions and reports two cases to disprove the contentions of the former.

In mentioning the work of James and MacDougal, the author takes issue with their idea that a kinesthetic idea must necessarily precede all voluntary motion, and in this contention, instances cases of children who suddenly walked a considerable distance without previously having tried to walk.

Speech, the author concludes, is a part of intelligence, for aphasia and apraxia are inseparable from other disorders of intelligence. The entire brain participates in the mechanism of speech, even the cerebellum. In considering the speech symptoms of dementia praecox, the author reviews Southard's work on the glioses in this disease and the similar work of Lambert in presenile psychosis, in both of which conditions the lesions are very diffuse.

In Chapter 7 the author considers elements in speech other than the use of words, i. e., tone and gesture which may convey a meaning quite opposite to that of the spoken word. These must be considered as integral parts of speech. No part of the brain or group of centers can be said to control speech, although the control of the motor part of speech may be localized in the area which controls the movements of the vocal cords and phonation.

In a final chapter the author catalogues other forms of speech difficulty existing in organic diseases—the “hot potato speech” of Friedreich’s ataxia, the monotonous, colorless speech of cerebro-cerebellar ataxia, paralysis agitans and Wilson’s disease, the scanning speech of multiple sclerosis and the slovenly, inadequate speech of the tabetic. The speech of paralysis agitans, Wilson’s disease and multiple sclerosis, the author considers to be due to a disturbance of the cerebellar mechanisms. After a review of the anatomy of these mechanisms, he concludes that the cerebellum coordinates and gives tone to speech as well as to any other motor phenomenon.

The author has given us an interesting and valuable contribution to the study of speech and its disturbances. His method of handling his material is rather original, and he makes the most of what he has. One could wish that he had more material, especially postmortem material which would have given additional conviction to his arguments. From his last paragraph one judges that the author feels that he has not proved his contentions as well as he would wish to or to the extent to which he believes them.

CASAMAJOR, New York.

CONTRIBUTION TO THE STUDY OF LUETIC-EPILEPSY. DIETZ
L. EDZARD, *Deutsch. Ztschr. f. Nervenhe.* 63: Nos. 5 and 6.

Epileptic manifestations following syphilitic infection present no particular difficulty when they are accompanied by indications which point to a syphilitic involvement of the brain or meninges. In those cases, however, in which a person infected with syphilis develops epilepsy without showing any specific syphilitic changes in the central nervous system, the problems of differentiation are more difficult and opinions more divergent.

Edzard presents in detail the study of the history of a man, 35 years of age, an artist of neuropathic heredity, who was subject to occasional epileptic attacks at infrequent intervals and who showed no evidence of mental deterioration. Alcoholic excesses and the hardships of a severe campaign as a soldier had no aggravating effect on the epilepsy. But in 1916 the patient contracted syphilis and within eighteen months he received three courses of intensive antisyphilitic treatment. Subsequent to the third course there was an increase in the epileptic attacks, which culminated in a status epilepticus. After subsidence of this condition marked mental deterioration manifested itself. Three months later a second status epilepticus developed, which was followed by mental hebetude, circumstantiality, irritability and disturbance of speech of a permanent character. The neurologic symptoms were transitory and the spinal fluid, except for a slight lymphocytosis detected in one examination, showed no deviation from the normal. The blood Wassermann test was at first negative, then it varied and finally became positive and remained so despite carefully exhibited treatment.

What bearing the syphilis had on the course of the epilepsy, and the explanation of the serologic findings, or rather, their paucity, is the problem Edzard discusses. He holds that the status epilepticus may account for the transitory lymphocytosis, but that in general there is no characteristic picture to be had in the spinal fluid in epilepsy.

Observations made on syphilitic persons without any history or indication of epilepsy, who after the infection develop epilepsy, seem to indicate that the epilepsy is of the symptomatic type. Redlich, on the other hand, explains the manifestations as organic but based on minute histologic manifestations.

Others hold that there is a toxicodynamic cause similar to that found in the parasyphilitic epilepsy of Fournier; Nonne speaks of a postsyphilitic epilepsy following late on syphilis, in which there is an increase and a stimulation of a latent epileptic disposition through the acquired syphilis.

In the present case the acute exacerbation and the progress of the epileptic symptoms may be ascribed to the toxic dynamic effect of syphilis. As is well known, recent studies have shown that in secondary syphilis there is frequently pronounced involvement of the nervous system. The connection, in the present instance, between the increase in the epileptic symptoms and the vigorous anti-syphilitic treatment must not be overlooked, and carries with it the suggestion that in the treatment of syphilis occurring in persons of the neuropathologic type great caution must be exercised and the application of a routine method avoided. In other words, there should be a greater individualization of therapy in syphilis of a neuropathologic type.

GOTTBRATH, San Francisco.

REEDUCATIONAL TREATMENT IN APHASIA. F. REICHMANN U. ED. REICHAU, *Archiv. f. Psychiat.* 60:8, 1919.

The optic-tactile method of reeducational treatment in aphasia is favored by these authors in both motor and sensory forms. The traumatic brain injuries of war, producing aphasia in otherwise normal brains, have afforded particularly desirable material for this study. They lay particular stress on the development of kinesthetic memory pictures to replace the usual auditory memory pictures, and claim to have achieved satisfactory results. A number of case histories are presented that give the technic of their method, which is in brief: The patient is shown the motor mechanism of the mouth, lips, etc., for each separate sound. His hand is placed over the larynx and then over the mouth and nose, in order to perceive the air vibrations. Then, by the aid of a mirror, under guidance of the instructor, the patient practices the formation of each vowel and consonant sound. The kinesthetic impressions of sound are thus formed later into syllables, words and sentences. A chapter is devoted to a discussion of the central nervous mechanisms concerned in recovery of speech. The practical experiences of the authors incline them to the view of a vicarious action of the right hemisphere. This view is supported by the clinical fact that patients who have learned to write with the left hand because of the paresis of the right, still favor the left hand after the paralysis on the right hand has cleared. Writing exercises with the left hand are said to influence favorably speech reeducation.

SCHALLER, San Francisco.

Society Transactions

CHICAGO NEUROLOGICAL AND CHICAGO PATHOLOGICAL SOCIETIES

Joint Meeting, April 14, 1919

HUGH T. PATRICK, M.D., *in the Chair*

TRANSPLANTATION OF PERIPHERAL NERVES. Presented by Prof. G. CARL HUBER, Ann Arbor, Mich.

The data to be presented have been gathered in the course of experimental work carried on in conjunction with the Division of Head Surgery of the Office of the Surgeon-General and extend over a period of somewhat more than one year. During this period Lieut.-Col. Dean D. Lewis, Majors J. F. Corbett and Byron Stookey, and Capt. T. Roberg have in succession received assignment to the department of anatomy, University of Michigan, where this work has been carried on. To their initiative and untiring and hearty cooperation, the progress of the work and the results attained owe much.

In the prosecution of the experimental operative work strict asepsis was followed. So far as possible, the surgical technic was that of modern aseptic surgery. In practically all of the experimental operations union was by primary intention.

Owing to the difficulty of obtaining catgut sutures of suitable size and quality for uniting resected nerves, we were early in our work led to adopt a silk suture and as technic developed, Triple 0, Corticelli silk thread, unraveled so as to obtain three fine threads, became the standard suture in nerve repair. Before use, the sterilized threads were thoroughly waxed with sterile wax; this facilitating greatly the application of the suture. In practically all of our experiments of nerve transplantation the silk suture was passed through the nerve trunk and nerve transplant. The passing of a silk suture through a nerve trunk and transplant is innocuous, the silk suture becoming early encapsulated in a thin layer of connective tissue which does not materially interfere with the downgrowth of central neuraxes.

The tissues obtained from the animals operated on have for the greater part been stained after the pyridin-silver method, and for many of the experiments were sectioned at close intervals, in cross and longitudinal sections, for the length of the nerve operated on. This method gives a differential staining for the neuraxes, especially desirable in a study of the regenerative processes of peripheral nerves.

Regarding nerve regeneration, we maintain that regeneration of a degenerated portion of a peripheral nerve is through downgrowth of new neuraxes derived from the central nondegenerated portion of said nerve, the peripheral degenerated portion taking no immediate part in the regeneration of the neuraxes.

In the earlier stages of this experimental work, and in association with Lieut.-Col. Dean D. Lewis, the question of injecting absolute alcohol into a living nerve, and related problems, was dealt with in four series of experimental observations. Though not coming immediately within the scope of

the question under consideration, they have a bearing on certain of the experiments to be considered. In one of these series the use of absolute alcohol to obviate the formation of amputation neuroma was tested.

Series No. 1.—Sciatic of Rabbits.—Thirty-three experiments; respective animals under observation for periods varying from two to 157 days. The sciatic of rabbits was exposed and injected in about the middle of the leg with about 0.5 c.c. of absolute alcohol, in two or three point injections. The sciatic was then cut from 3 to 5 mm. distal to the point of injection and from 1 to 1.5 cm. of the distal sciatic stump resected. The wound was then closed. The operated animals were killed at intervals and it was observed that this simple procedure obviated the formation of amputation neuroma.

Series No. 2, a Control Series.—In this series it was ascertained that amputation neuromas form after every section of a peripheral nerve, even though the section be made under strict asepsis, with immediate closure of the wound and with wound healing by primary intention. In central nerve stumps injected with absolute alcohol, exposed and examined at stated intervals, it was noted that such central stumps terminate in a fine tapering end with no appreciable enlargement. From four to five weeks after section and alcohol injection, fine, newly formed neuraxes penetrate these tapering ends and may even grow beyond, but these down-growing neuraxes do not present the tangle and excessive proliferation of new nerve fibers noted in an amputation neuroma and there is primarily no proliferation of sheath cells. For use in human surgery it is recommended that from 1 to 2 c.c. of absolute alcohol be injected into the distal end of the nerve stump, from 1 to 2 cm. central to the cut end of the nerve. It is suggested, especially for the larger nerves, that a series of point injections be made, perhaps in two or three tiers, so that all parts of the nerve be reached with absolute alcohol. This method can be recommended as a simple and efficient method for preventing the formation of amputation neuroma.

Series Nos. 3 and 4.—In a further series the behavior of a living nerve injected in course with absolute alcohol or acetone, and without cutting the nerve, was experimentally studied. The injection of absolute alcohol into a living nerve causes fragmentation of neuraxes and myelin in the field coming under the immediate influence of the absolute alcohol. This fragmentation differs from that of Wallerian degeneration and is not accompanied by a primary proliferation of sheath cells. In due time, regeneration by the down-growth of central neuraxes follows. In cases of severe causalgia, in which section of the nerve is contemplated, injection of absolute alcohol without nerve section should be considered.

The great majority of our experiments have dealt with the question of bridging nerve defects of such extent that the severed nerve ends, after the necessary repair and resection, cannot be brought end to end, without undue tension, by process of simple suture. In this connection the question more immediately under discussion, namely, nerve transplantation, received especial consideration. In the series of experiments listed, the following nomenclature is used: A segment of nerve used to bridge a defect in a peripheral nerve, taken from another nerve of the same individual, is designated an *auto-nerve-transplant*; a segment of nerve taken from another individual, but of the same species, is designated a *homo-nerve-transplant*; a segment of nerve taken from another individual and of a different species is designated a *hetero-nerve-transplant*. Collateral questions, such as the use of certain membranous structures for purposes of sheathing a nerve-transplant and suture lines; the

question of the use of tubular sutures in nerve repair and of nerve suture under undue tension, subjected to experimental test, are not reported on at this time.

Experimental observations warrant the conclusion that bridging nerve defects by means of nerve transplants is a legitimate operation and one to be recommended in cases where there is loss of nerve substance of such extent that ordinary suture cannot be made without undue tension.

The experimental observations dealing with nerve transplantation and related questions are here considered under the following series:

Series No. 5. Auto-Nerve-Transplants.—Sciatic of dogs. Seventeen experiments, with respective animals under observation for periods varying from eleven to 382 days.

In these experiments the sciatic nerve was exposed and resected to the extent of from 2.5 to 3 cm. The resultant defect was bridged by means of a segment or several segments of nerve taken from one of the sensory cutaneous branches of the same dog. In our experiments the transplant was taken from the cutaneous radial branch. Since in surgical practice in dealing with the repair of the larger nerves, the resection of a normal nerve of approximately the same size for purposes of obtaining an auto-nerve-transplant is not justified, it has been the procedure to use for an auto-nerve-transplant a segment of one of the cutaneous nerves of much smaller size than the nerve to be bridged. The resultant difference in the size of the nerve to be bridged and the transplant has mitigated against obtaining optimum results. It occurred to us that this difference in size of transplant and nerve to be bridged might in a measure be overcome by making use of several segments of a smaller nerve to bridge a defect in a larger nerve. This procedure we designate a cable-auto-nerve-transplant. In eight experiments out of the seventeen of this series this method was tested by taking four segments of the cutaneous radial nerves to bridge a defect in the sciatic. These segments were sutured separately or in groups of two, both centrally and distally, between the resected ends of the sciatic. In the remaining nine experiments of this series the defect in the resected sciatic was bridged by using, respectively, one segment of the cutaneous radial, five experiments; two segments, three experiments; three segments, one experiment.

The observations made on these experiments may be summarized by stating that the "cable auto-nerve-transplant" presents a method for bridging nerve defects which gives every promise of favorable results. Within a few days after the operation the several segments of nerve transplanted become surrounded by connective tissue, so as to form an epineural sheath, binding them together in one nerve trunk in which the funicular arrangement of the several nerve segments transplanted is fully maintained. Downgrowing neuraxes coming from the central stump penetrate and pass through the several funiculi to reach the distal segment which in time becomes penetrated by new neuraxes. These experiments have been carried on for a time of sufficient length to obtain new motor nerve endings in the calf and plantar muscles and evidence of sensory regeneration. It is admitted that this operation is tedious, requires care and some skill and necessitates the making of a second wound. However, the experimental results justify its recommendation. In surgical practice the cutaneous radial and the cutaneous portion of the musculo-cutaneous of the arm, the lesser sciatic, and especially the sural of the leg may at convenience be selected as the nerves from which segments for a cable auto-nerve-transplant may be taken.

Series No. 6. Homo-Nerve-Transplants.—Sciatic of rabbits: seven experiments, with respective animals under observation for periods varying from eight to eighty-three days.

In these experiments a segment from the sciatic of one rabbit was used to bridge a defect in the sciatic of another rabbit experimentally resected. These experiments show that a fresh homo-nerve-transplant may be employed to bridge a nerve defect with every promise of success. Since chance alone would permit this operation in surgical practice other experiments dealing with homo-nerve-transplants were devised and will be considered in this report.

Series No. 7. Hetero-Nerve-Transplants.—Sciatic of rabbits: thirty-nine experiments. In sixteen of these the two sciatics of half grown guinea-pigs were used to bridge a defect in the sciatic of grown rabbits; in twenty experiments one of the sciatics of a full grown guinea-pig was used to bridge a defect in the sciatic of a full grown rabbit; in three experiments one of the major nerves of a dog was transplanted to a sciatic of a rabbit. The respective animals were under observation for periods varying from three to over 300 days.

The question of the possibility of using hetero-nerve-transplants is of academic and practical interest. The ease with which fresh, normal nerves may be obtained as hetero-nerve-transplants warrants the interest taken by surgeons in experiments dealing with hetero-nerve-transplants. Opinions current in literature are not unanimous as concerns experimental observations on hetero-nerve-transplantation. Our own results may be summarized in this brief abstract in the statement that neurotization of the distal stump through a hetero-nerve-transplant is experimentally possible. However, the results obtainable are not as certain and not as favorable as when auto-nerve-transplants or homo-nerve-transplants are used, and the resultant distal regeneration not as complete. Therefore, this procedure cannot be recommended as an operation of choice in surgical practice.

The following series of experiments deals with the use of degenerated auto-nerve-transplants, homo-nerve-transplants and hetero-nerve-transplants. Every nerve transplanted undergoes degeneration. Whether nerves degenerated before transplantation would prove more favorable for downgrowth of central neuraxes than nerve transplants taken from fresh, normal nerves which would undergo degeneration after transplantation, seemed worthy of experimental test.

Series No. 8. Degenerated Auto-Nerve-Transplants.—Sciatic of dogs: three experiments; respective animals under observation for periods varying from 127 to 416 days.

In this series of experiments the sciatic nerve was cut and the wound closed. About twenty days later the cut sciatic was again exposed and a segment of the degenerated distal stump transplanted to the resected ulnar of the same dog. The experiments of this series are too few to warrant drawing definite conclusions. The statement seems justified that regeneration of the distal stump of a resected nerve through a degenerated auto-nerve-transplant is possible, but that such regeneration is not more favorable nor more rapid than when a nondegenerated auto-nerve-transplant is used.

Series No. 9. Degenerated Homo-Nerve-Transplants.—Sciatic of dogs: five experiments; respective animals under observation for periods varying from seventeen to 328 days.

For this series the sciatic nerve of a number of dogs was cut and the wound closed. Some twenty to thirty days later the cut sciatic was again exposed and a segment taken from the degenerated distal stump and transplanted to bridge

a defect in a resected sciatic of another dog. The observations made on these experiments warrant the general conclusion that regeneration of the distal stump of a resected nerve may be obtained through a degenerated homo-nerve-transplant. The possibility of using a degenerated nerve as a homo-nerve-transplant in surgical practice is warranted by these observations.

Series No. 10. Degenerated Hetero-Nerve-Transplants.—Sciatic of rabbits: eighteen experiments; respective animals under observation for periods varying from three to 244 days.

For these experiments one of the major peripheral nerves of a dog was cut and allowed to degenerate for periods varying in the respective experiments from about twenty to thirty days. A segment of the degenerated nerve was then transplanted to the resected sciatic of a rabbit. The syncytial, nucleated strands found within the sheaths of degenerated peripheral nerves, having undergone Wallerian degeneration, may be regarded as simulating embryonic tissue. Such tissue, it was argued, should prove more favorable for downgrowth of central neuraxes than would be the nondegenerated nerve fibers of a hetero-nerve-transplant. The observations made on this series of experiments do not bear out this assumption. It was observed that the degenerated hetero-nerve-transplant undergoes a secondary degeneration. In certain of the experiments downgrowth of the central neuraxes through and on the degenerated hetero-nerve-transplant was obtained. However, the results, even in the experimental observations, were so uncertain that the adoption of this procedure in surgical practice is not warranted.

The following series of experiments was devised to test the possibility of storing homo-nerve-transplants for a period of several weeks before use. The possibility of obtaining human nerves under aseptic precaution, from amputated limbs, and storing them until operative procedure demands their use, would obviate the difficulty experienced in surgical practice of obtaining fresh human nerves on demand.

Series No. 11. Homo-Nerve Transplants Stored in Petrolatum.—Sciatic of rabbits: eight experiments; respective animals under observation for periods varying from sixty-six to 155 days.

Dujarier and Francois (*Bull. et mém. Soc. de chir. de Par. 44*: January, 1918), in a series of twenty-four cases reported the use of homo-nerve-transplants stored in petrolatum before use in operative procedure. They recommend that the nerves be removed from amputation stumps under aseptic precautions, and placed at once in warmed, sterile petrolatum and then kept at nearly 0 C. temperature. Nerves were thus kept for several weeks. Before use, the petrolatum was again warmed, the nerve removed and washed in warm sterile serum. Twenty-four cases were operated on and reported before sufficient time had elapsed to enable determining the ultimate results. It is reported that in all cases but one the healing of the wound was by primary intention.

At the time our experiments were undertaken we were unable to find any experimental observations on this method. In our experiments the method suggested by the French observers was followed as closely as the brief account descriptive of the method permitted. The sciatics of full grown rabbits were removed under asepsis, placed at once in sterile petrolatum contained in tubes warmed to the melting point of the petrolatum. The tubes were then placed in a small ice chest kept at constant temperature of 3 C. In the several experiments, after periods varying from four to thirteen days, the nerves thus treated were used to bridge defects of about 3 cm. lengths, caused

by resection of the sciatics of rabbits. The results of these experiments are gratifying. Functional return was noted in the experiments of longer duration. Time has not permitted a histologic study of the tissue removed. In the experiments of shorter duration a microscopic study of the transplant shows abundant down growth of central neuraxes through the transplant. The experimental observations warrant the statement that this method, as suggested by the French observers, deserves consideration in surgical practice.

Series No. 12. Homo-Nerve-Transplants Stored in Liquid Petrolatum.—Sciatic of rabbits: forty experiments; respective animals under observation for periods varying from one hour to 229 days. (To date, certain of the animals are still under observation.)

Under the method of storing nerves in petrolatum (Series No. 11) it is necessary to warm the petrolatum to the melting point before the nerve segment can be placed therein, likewise when the nerve segments are removed therefrom. To obtain sterile serum is not always easy. For these reasons we have devised a simple method which consists in storing nerves in liquid petrolatum. We have used Squibb's liquid petrolatum. This is a clear, bland fluid. The required quantity was placed in large tube-vials, corked with cotton plugs, then autoclaved on successive days. After cooling to room temperature, the tubes with liquid petrolatum were placed in the ice chest and cooled to 3 C. The sciatics of full-grown rabbits were removed with asepsis, placed in the cooled and sterile liquid petrolatum and kept at 3 C. Nerve segments thus stored in liquid petrolatum were used in the several experiments after storage of from four to thirty-nine days, as nerve transplants to bridge defects of approximately 3 cm. length in the sciatic of rabbits. For each experiment the tube containing the nerve transplant was taken from the ice chest just before the operation, and when required, the nerve transplant was taken from the liquid petrolatum, and this allowed to drain off by holding the nerve segment suspended from one end by means of forceps. Sutures were then placed near each end of the nerve segment and the ends cut by means of sharp scissors to within about 2 mm. of the sutures. The nerve segment was then sutured to the two ends of the resected sciatic. Nerves stored in this manner even for a period of thirty-nine days, retain their color and consistency remarkably well. The experimental animals of this series of longer duration, six months and over, have not as yet been killed and examined. The experiments completed and fully studied warrant the conclusion that neuraxes derived from the central end will grow through a homo-nerve-transplant, having a length of 3.5 cm., and stored in liquid petrolatum for a period of somewhat over five weeks, quite as well as through a freshly removed homo-nerve-transplant. Very satisfactory neurotization of the degenerated distal segment has been obtained through a homo-nerve-transplant stored in liquid petrolatum, having a length of somewhat over 3 cm., 100 days after the operation. Experimental observations warrant the statement that the use of human nerves obtained from amputated members and stored in liquid petrolatum as here directed, for the purpose of bridging nerve defects, deserves serious consideration as a surgical procedure.

Series No. 13. Homo-Nerve-Transplants Stored in Sterile 50 Per Cent. Alcohol.—Sciatic of rabbits: eighteen experiments, with respective animals under observation for periods varying from two to 151 days. (Certain animals of this series are still under observation.)

Nageotte (*Bull. et mém. Soc. de chir. de Par.* 44: 1918) has recommended the use of nerve transplants stored in 50 per cent. alcohol. For this series

the sciatics of full-grown rabbits were removed under asepsis and placed at once in 50 per cent. alcohol contained in wide-mouthed glass-stoppered bottles. In the 50 per cent. alcohol the nerve segments were stored for periods varying in the several experiments from seven to twenty-nine days. Just before use as a nerve transplant the nerve segment was taken from the 50 per cent. alcohol and placed in a dish containing sterile normal salt solution in which they remained from fifteen to twenty minutes. When taken from the 50 per cent. alcohol the nerve segments are quite hard, though not brittle. After a short stay in the normal salt solution they again become quite pliable. The nerve segments were taken from the normal salt, sutures placed, the ends freshened by cutting with sharp scissors about 2 mm. from the suture lines and the operation completed by transplanting the nerve segment into a defect caused by resection of the sciatic of a rabbit. Time has not permitted conclusive observations on this series. The observations made thus far indicate clearly that downgrowing neuraxes coming from the central stump penetrate readily a homo-nerve-transplant having a length of 3 cm. and stored in 50 per cent. alcohol for nearly four weeks, and thus guide the downgrowing neuraxes to the degenerated distal stump.

In this series of experiments of homo-nerve-transplants stored in petrolatum, liquid petrolatum, or 50 per cent. alcohol, the observations indicate that the nerve segments thus stored do not retain a latent viability. The sheath cells of the transplants show no evidence of proliferation and appear not to have a biologic significance. The neurolemma sheaths of the transplanted nerve fibers are well preserved and through these the downgrowing neuraxes of the central stump find ready paths and thus reach the distal stump. Therefore, it seemed desirable to test the behavior and the value of hetero-nerve-transplants stored in liquid petrolatum and 50 per cent. alcohol and for this purpose the following series of experiments were undertaken.

Series No. 14. Hetero-Nerve-Transplants Stored in Liquid Petrolatum.—Sciatic of rabbits: six experiments, respective animals under observation for periods varying from five to 117 days. (Certain of the animals of this series are still under observation.)

For this series certain of the major nerves removed from dogs under strict asepsis were stored in liquid petrolatum, as described under series No. 12, for periods varying from eleven to twenty-five days, and then used as nerve transplants to bridge defects in the sciatic of rabbits caused by resection. None of the longer time experiments of this series have thus far been studied histologically. Therefore, no conclusions as to the value of this method can at the present time be drawn.

Series No. 15. Hetero-Nerve-Transplants Stored in 50 Per Cent. Alcohol.—Sciatic of rabbits: three experiments, respective animals under observation for periods varying from sixty-four to 117 days. (Two of the animals are still under observation.)

For this series certain of the major nerves of dogs were removed and stored in 50 per cent. alcohol as described in Series No. 13. They were then used to bridge defects in the sciatic of rabbits. Two of the animals of this series are still under observation; the other experiment was terminated by death of the animal too soon after operation to admit of drawing definite conclusions.

References are frequently made in surgical literature to the use of certain membranous structures employed as sheaths which are wrapped about nerve transplants or suture lines. Concerning the necessity or value of such sheaths

there is no unanimity of opinion. The following series of experiments was undertaken to test their value. It should be stated that it is here recognized that the clean, surgically aseptic, operative wounds in animal experiments, made through normal tissue, do not simulate the operative wounds in surgical practice in many cases of secondary nerve suture, made in the presence of abundance of cicatricial tissue. However, it was hoped that certain general principles might be ascertained to serve as guides in surgical practice.

Series No. 16. Auto-Nerve-Transplant with Nerve Transplant and the Suture Lines Wrapped in Several Layers of Cargile Membrane.—Sciatic of dogs, thirteen experiments, with respective animals under observation for periods varying from twenty hours to 359 days.

In these experiments we made use of Cargile membrane prepared by Johnson & Johnson. The left sciatic and the right ulnar nerves of respective dogs were exposed and a segment of the right ulnar from 3 to 4 cm. long was transplanted to bridge a defect in the sciatic, caused by resection. After suturing the transplanted ulnar, the transplant and the sutures were surrounded by two or three layers of Cargile membrane, closely applied and wrapped about the nerve. The wound was then closed. Untreated Cargile membrane, as here used, it was found, was absorbed within a period of ten days; that is to say, before it could serve a purpose in preventing connective tissue formation in the immediate vicinity of the transplant and suture lines. Therefore, its value in surgical practice is seriously questioned.

The difficulty of obtaining Cargile membrane at the time these experiments were made led us to attempt resterilization of that portion of each membrane not used at any one operation. This was accomplished by placing the same in 70 per cent. alcohol in which they were stored, often for days. Before their use they were placed several hours in absolute alcohol and before use in operations taken from the absolute alcohol, spread out on dry sterile towels and allowed to dry. In five of the thirteen experiments of this series such alcoholized Cargile membranes were used to wrap the nerve transplant. It was found that this very simple method very materially alters the absorbability of the Cargile membrane as found in the market. In one experiment terminated nearly five months after the operation distinct evidence of the Cargile membrane was found. In another experiment terminated nearly seven weeks after the operation the alcoholized Cargile membrane was found to have been practically unaltered and remained closely wrapped about the nerve. There was evident no material increase of connective tissue about the alcoholized Cargile membrane thus used as a sheath.

The use of alcoholized Cargile membrane, prepared as above stated in double or triple layers, as a sheath for wrapping nerve transplant or suture lines after nerve suture, when such sheathing is deemed necessary, deserves consideration in surgical practice.

Series No. 17. Auto-Nerve-Transplant Wrapped in Auto-Fascial Sheath.—Sciatic of dogs, fifteen experiments, with respective animals under observation for periods varying from fourteen to 324 days.

In this series of experiments the left sciatic and right ulnar, or vice versa were exposed and a segment of the ulnar transplanted to the sciatic. After suturing the transplant in place this was wrapped in a piece of fascia recently taken from the same dog. The fascial membrane taken was removed and trimmed to such size that when applied it extended about 8 mm. beyond the central and distal suture lines of the transplant, and when wrapped about the nerve formed a closely fitting tube with edges overlapping about 5 mm.

Fine silk stay sutures and several half mattress sutures were placed to hold the sheath in position and complete the tube. The experiments of this series admit the general conclusion that an auto-fascial sheath is very slowly absorbed, evidence of its persistence having been observed three months after operation. However, even in the aseptic wounds made in normal tissue, where use could be made of the fascial planes for exposing the nerves, there is observed a distinct connective tissue proliferation about the fascial sheath, to such extent as to prejudice against this procedure in surgical practice, especially in operations where nerve repair is made through and in cicatricial tissue.

Series No. 18. Auto-Nerve-Transplant with Nerve Wrapped in a Formalinized Arterial Sheath.—Sciatic of dogs, eight experiments, with respective animals under observation for periods varying from six to 241 days.

In this series of experiments the left sciatic and right ulnar, or vice versa, were exposed and a segment of the ulnar transplanted to bridge a defect in the resected sciatic. After the transplant was sutured in place, this was surrounded by a sheath prepared from the wall of a formalinized artery. The carotid arteries of large dogs were removed and stretched over glass rods of suitable size. They were then fixed in 5 per cent. liquor formaldehydi for forty-eight hours; washed in water twenty-four hours; boiled in distilled water for twenty minutes; then stored in 70 per cent. alcohol in sterile, wide-mouthed, glass-stoppered bottles for from several days to several weeks, as necessity demanded. Before use, a segment of the fixed artery of required length was slipped from the glass rod, cut longitudinally along one side and placed in sterile normal salt solution for about thirty minutes. The arterial sheath thus prepared was then wrapped about the nerve transplant and allowed to extend over the suture lines about 5 mm. and fixed in place by central and distal stay sutures and several half mattress sutures, using fine silk. The experiments of this series admit of drawing the following general conclusion: that a formalinized arterial sheath, prepared as above stated, remains in place and without absorption for several months, and this without inciting material increase of connective tissue. It is more particularly the elastic tissue of the vessel wall that resists absorption. Since formalinized arterial sheaths are easily prepared and may be kept on hand in sterile condition in 70 per cent. alcohol, and are easily applied, this method deserves consideration in surgical practice, when sheathing of a transplant or suture line in nerve repair is deemed desirable.

Series No. 19. Auto-Nerve-Transplant with Completely Detached Auto-Fat Sheath.—Sciatic of dogs, two experiments; animals under observation, one, four days; the other, 324 days.

In this series the left sciatic and right ulnar were exposed and a segment of the ulnar transplanted to the sciatic. The nerve transplanted and the suture lines were then wrapped in a membrane of subcutaneous fat, taken from the same animal to one side of the sciatic wound. After removing the membrane of fat, which had an average thickness of about 5 mm., this was gently washed in sterile normal salt solution, so as to remove as far as possible the adherent blood, and was then wrapped about the nerve transplant and suture lines. The sheath was held in place by the use of several fine silk stay sutures. One of these experiments was terminated by the death of the animal four days after operation; in the other, the animal was killed 324 days after operation. In the latter experiment, regeneration of the distal segment was obtained. However, the region of the transplant was surrounded by a distinct layer of dense fibrous tissue occupying the region of the fat sheath and binding the trans-

plant to the subcutaneous tissue. Definite conclusions based on this single experiment hardly seem warranted. However, the result obtained argues against the use of a complete detached fat sheath, even when this is taken from the same animal, since the fat membrane is replaced by dense fibrous tissue.

Series No. 20. Tubular Suture by Use of Formalinized Artery.—Ulnar nerve of dogs, twelve experiments, respective animals under observation for periods varying from four to 298 days.

In this series we made use of the resected ulnars of certain of the experiments in Series Nos. 16 to 19, to test experimentally the value of an arterial tubular suture as recommended by Foramitti and since used in the Russo-Japanese war by Hashimoto. The formalinized arterial tubes were prepared as stated under Series No. 18, essentially as described by Foramitti. Before use, the formalinized carotid artery of a large dog was taken from the glass rod and a segment about 1 cm. longer than the defect in the nerve to be repaired was removed and placed for about one-half hour in sterile normal salt solution. A fine silk suture armed with a fine needle at each end was then passed through the central and distal stump of the resected ulnar, about from 2 to 3 mm. from the cut ends. The needles of each suture were then passed through the opposing sides of the formalinized arterial tube from 7 to 8 mm. from the respective ends of the tube and the ends of the resected nerve, central and distal, drawn into the lumen of the arterial tube and held in place by knotting the silk sutures over one side of the arterial tube. The wound was then closed by using the necessary fascial and skin sutures. In this series of experiments no nerve-transplant was inserted, the ends of the resected ulnar, from 4 to 5 cm. apart, were merely inserted into the ends of the arterial tube and kept in place by means of stay sutures.

In this series, as stated for series No. 18, it was found that the formalinized artery resists absorption for a period of at least three months. In certain of these experiments it was evident that one or the other of the ends of the nerve had been pulled out of the lumen of the arterial tube some time soon after the completion of the operation. In two of the experiments of more than three months' duration neurotization of the distal ulnar was obtained through downgrowing neuraxes of the central stump, conveyed to the distal stump through the lumen of the formalinized artery. This series confirms the possibility of obtaining regeneration of the distal degenerated end of a nerve, after loss of from 4 to 5 cm., through a tubular suture in case the tubular suture resists absorption for a period of sufficient length to admit of downgrowth of the central neuraxes to the extent of reaching the central end of the degenerated distal stump. However, the method cannot be recommended for adoption in surgical practice since other methods for bridging nerve defects offer greater assurance of success.

Series No. 21. Direct Suture of Nerves Under Tension.—Ulnar nerve of dogs, eleven experiments, respective animals under observation for periods varying from twenty-two to 324 days.

In this series use was made of the resected ulnar nerves of certain of the experiments in Series Nos. 16 to 19. A silk suture was passed through the central and distal stumps of the ulnar, resected to the extent of from 4 to 5 cm. and by applying tension and flexing the limb the resected nerve ends were brought to as close approximation as possible and sutured. In a number of these experiments the line of suture was wrapped with several layers of alcoholized Cargile membrane or a formalinized artery sheath was applied. In other experiments the wound was closed without sheathing the

suture line. Purposely, no endeavor was made to immobilize the limb. On opening the wounds in the several experiments at stated intervals, it was noted that in all but one experiment the suture had torn out either centrally or distally and that the nerve ends had separated for a distance about equal to that obtained before the tension sutures were applied. In the one experiment in which the suture did not appear to have been torn out, limited neurotization of the distal stump was obtained.

DISCUSSION

LIEUT.-COL. DEAN D. LEWIS: We are to be congratulated on having this paper presented at the present time when so many peripheral nerve injuries are being treated. There is one thing that I think has been definitely demonstrated; namely, that all regeneration takes place from the proximal stump. I also believe that it has been definitely proved that without protoplasmic bands no regeneration takes place. That is illustrated in section of the spinal cord or in section of the optic nerve.

I believe all peripheral nerve surgery should be taken care of in evacuation hospitals and that in all cases primary suture should be done. It is exceedingly interesting to study peripheral nerve injuries in the evacuation hospital, say fifteen hours after injury. In all the cases I have seen, either from high explosives or machine-gun bullets, the nerves immediately after section have been found contused. A machine-gun bullet may make a perfectly clean hole, but if one examines the peripheral nerves, one finds on each side of the defect for half an inch that all elements of the nerve are destroyed. So if an attempt is made to make suture, considerable approximation must be accomplished to get neurofibrilla in contact with neurofibrilla. In the musculospiral it is practically impossible to approximate these without flexion of the elbow, or in the sciatic without flexion of the knee to a right angle.

There has never been much opportunity to study these nerves that had primary suture. It is unfortunate that the primary sutures at the evacuation hospitals are so distributed in this country that it is impossible to make a study. At Fort Sheridan I have found only one case of extensive primary suture. Quite a number of the cases, in fact the majority of them, have had a separation of the nerve ends with a neuroma. They have had complete loss of function primarily, but they have gradually improved. Now, six or seven months after the injury, we find that this preliminary return of function has come to a stationary point. I operated on two cases this morning, which had improved rapidly for some time, but for the last three months had been stationary. In one of these cases, which was the sciatic, I found just a few adhesions. The only thing to do was to excise the neuroma and attempt a neurolysis and suture the healthy muscle above and below and wait for return of function. The second case I operated on this morning had what was theoretically a complete ulnar lesion and a partial median lesion. The ulnar had a very distinct lesion of the nerve with some of the funiculi destroyed and yet there were enough funiculi left to conduct the impulses through. The median was bound in scar tissue. It was merely an indication for neurolysis. There are very many cases with division of both nerves. The most serious are the lesions in the arm in which the median and ulnar are divided. It is surprising that an ulnar lesion alone is less frequent than an ulnar combined with a median. If you flex the forearm to get an end-to-end suture of the separated ends of the ulnar, it is difficult to handle the

median. It is much better to make an end-to-end suture of the median and to transplant in the ulnar than to sacrifice the ulnar by doing some other operation on the median.

There is no doubt that the ideal nerve repair is end-to-end suture and that it should be attempted in all cases. To resect the humerus in order to make an end-to-end suture of the median or the ulnar, in my opinion, should never be done.

In lesions of the sciatic of over $2\frac{1}{2}$ inches' defect, one can flex the knee to a right angle and immobilize and close a defect of nearly $2\frac{1}{2}$ inches. In the case of the ulnar just above the wrist-joint, it is practically impossible to close any defect by flexion of the wrist. In these cases an auto-transplant ought to be used. In most of these cases you can get an auto-transplant directly from the site of your operation. For instance, in the case of the median or ulnar, you can use the cutaneous branch. I think that is the operation of choice. In cases where you cannot well sacrifice the cutaneous nerve, I think a man is justified in using some form of tubulization, using either a formalinized artery or a fascial tube. A fascial tube has a distinct limitation in all these injury cases, because a fascial tube transplanted in the presence of scar tissue will become scar tissue. That is the fate of fascial tubes in all these wounds.

I think we have three things to take into consideration. First, primary end-to-end suture on those cases with small nodules in the nerve. Then linear section and auto-neurolysis with the idea of allowing the nerve to expand to allow the nerve axons to grow through. Second, combined with this, the operation in which one dissects off the scar tissue. Third, if one cannot bridge the defect, an auto-transplant should be used.

In all these nerve operations we must consider the after-treatment. There are some sad things happening in war surgery by over-emphasis of certain points. Thomas, years ago, said that all paralyzed muscles should be placed at rest. We heard for many years about the cocked-up splint for musculo-spiral paralysis. Now, some of these boys have worn the cocked-up splint for the cure of drop-wrist for so long that they have an ankylosis of the wrist. It is just as harmful to over-stretch healthy muscles as it is to maltreat paralyzed muscle. To make a primary suture is by no means to finish the case. The after-treatment is most important in the correction of the paralysis.

Another thing sometimes forgotten is that up to the present time we have not had the opportunity of studying the results of our operations on nerves. I do not believe we can say anything about the return of function in these cases, even in the musculo-spiral or popliteal, for at least seven months. It would be very unfortunate if any of these wounded men were discharged before the period has expired in which we can expect a regeneration, because if we are going to learn anything in the line of peripheral nerve surgery from this war, these men must be watched for at least two years before we can make definite statements in regard to operations on peripheral nerves.

MAJOR LEWIS J. POLLOCK said that statistics relative to the incidence of peripheral nerve lesions sustained in battle were not as yet available. Those obtained from reports of the American Expeditionary Forces were not reliable for the reason that many cases of peripheral nerve lesions were not referred to the neurologist. As a result a large number of patients suffering with severe wounds, or those requiring complicated dressings were not observed from the neurologic standpoint. As consultant to a number of base hospitals, Major Pollock had between thirty and fifty such cases referred to him during a

month from each of these hospitals. A survey made of the cases brought to his attention on Sept. 7, 1918, when the population of the hospital under investigation was 1,440, revealed forty cases, or a percentage of 2.77.

In the early part of November, 1918, the total figures submitted to the chief consultant in neuropsychiatry led him to believe that the percentage of peripheral nerve cases in the American army was 1.76. This did not approach the figures given by the French and British, which were nearly 6 per cent. At this time a bed to bed examination of all patients wounded in extremities was made and in four hospitals under Major Pollock's supervision it was found that of 2,130 cases examined, 332 had peripheral nerve lesions. The total population of these hospitals at the time was 7,050. The percentage of peripheral nerve lesions in the total population was 4.5. The percentage of peripheral nerve lesions in cases examined was 14.9.

Analyses of the types of lesions seen were inaccurate for similar reasons. The prominence of certain clinical symptoms in some injuries made it necessary to submit such a case to the neurologist, so that ulnar lesions were very frequently referred to the neurologist whereas median lesions were frequently overlooked. Internal popliteal lesions were rarely referred to the neurologic department.

The lack of standard nomenclature often contributed to the reports of internal popliteal lesions where a partial sciatic lesion was present.

The striking feature of the clinical picture of early nerve lesions was the large percentage of marked and rapid improvement. An analysis of the first 100 cases seen in Base Hospital No. 13 showed that after three months sixty-one cases had been discharged. Twenty of these cases had sufficiently recovered to be sent to convalescent and replacement camps. Thirty-nine cases remained in the hospital; twenty of these were manifestly partial lesions, and only five were so severe as definitely to indicate the necessity for surgical interference.

The actual percentage of recoverable cases of peripheral nerve lesions can only be ascertained when accurate statistics of the cases suffering with disability as the result of a peripheral nerve lesion may be compared to statistics of the occurrence of peripheral nerve lesions in battle. It might be mentioned that probably 10,000 peripheral nerve lesions were sustained in battle. Not very many more than 3,000 cases had been classified as peripheral nerve cases in the hospitals in the United States. Only one third of the cases observed in United States General Hospital No. 28, Fort Sheridan, Ill., would require operative interference and of these cases but the smaller proportion would require nerve suture.

DR. W. R. CUBBINS: I would like to ask the essayist the age of the animals that were used and if the transplants were put into nerves that were freshly sectioned or into nerves that had been traumatized.

DR. A. B. KANAVAL: I would like to ask one question: In the transplantation of tissue from one person to another is it not a good plan to test the cytolytic action of the serum of the donor on the serum of the recipient of the transplanted tissue? In man it may differ from that of the lower animals. In man we more often have a cytolysis that affects the cells of the transplant. Before I went to war I was engaged in the experimental investigation of transplantation and we had very great difficulty to obtain dogs in which we could show hemolysis.

As to the combined injury of the ulnar and median: When there was a defect in the ulnar that was difficult to bridge, I transplanted the ulnar to the

anterior surface of the joint thus, placing the nerve on the flexor instead of on the extensor surface. In that way I have been able to treat both lesions.

PROFESSOR HUBER (closing): In all these experiments except the first few in the treatment of neuroma, practically all the dogs that were used were 2 or 3 years old. I have only two experiments of secondary transplantation in which the neuroma was cut out and transplant put in. One of these happened to be a homo-degenerated nerve and the other was a homo-transplant. Personally, I feel that it makes no difference whether we are operating on fresh animals or not. I think in all this work we are simply gaining practice that can be applied to surgical technic. I think one could remove a neuroma not only once but several times, and the neuroma will reform and reform. I think the surgeons have had that experience with their methods in the past, and have found that neuromas reform after they have been removed. That means, of course, a new outgrowth from the stump. I cannot say how many times that will take place, but I know it will take place a number of times. I think as far as the neuromas are concerned that they will grow down from the stump after the primary injury. There is a little doubt as to how long after degeneration the nerve fibers will go down into the peripheral stump. I think Colonel Lewis collected statistics for a time, and I think he found that after the ninth month this occurred, but up to the ninth month it made very little difference as to the time that elapsed between the operation and the suture. After the ninth month there was a difference in the result, although, as I recall, there were successful cases in which the suture was done years later.

I feel that the transplant may be either an auto, hetero, or one of the homo sort in case of secondary suture if enough of the stump is removed to get above the neuroma into the healthy tissue. The neurons will grow just as they did primarily. If there is no transplant, there will be formed a new neuroma.

I am aware of the situation as far as human surgery is concerned with reference to hemolysis and the other condition that Dr. Kanavel speaks of. I do not think it is necessary to have a living transplant. I think stored transplants show that qualification. I was myself surprised to see neuraxes growing down in the transplant after it had been soaked in 50 per cent. alcohol, and still they grow down in a nerve that has been injected with absolute alcohol, as is done in cases of neuralgia. I suppose even Dr. Patrick has had cases of recurrence of the neuralgia after injections of alcohol.

DR. PATRICK: It *always* returns.

DR. HUBER: There is very good experimental evidence. I believe that the neuron sends out neuraxes which make use of the neurolemma sheaths as paths of least resistance and reach the peripheral stump in that way. I want to take this question with methods other than the silver staining. I think some time, if time permits, I shall study again nerve regeneration in the central nervous system and optic nerve. I am not quite certain whether the opinion of surgeons and experimenters in regard to regeneration in the central nervous system and in the optic nerve is correct, and whether after all there is not an attempt at regeneration. We have in the central nervous system and in the optic nerve no neurilemma sheaths. We have no paths of least resistance, no little tubes through which the axons can pass. We have neuroglia which is very quickly replaced by connective tissue. I believe one will be able to show that if the central root is cut and sutured, the neuraxes will pass up

into the cord so far as there are neurolemma sheaths, and then form a neuroma inside as soon as you get into the tissues of the central nervous system. I do not recall any very recent work. I do not recall any careful experimental work more recent than that of Ströbe, with the technic of whose work I am familiar. I used it in 1895, and since that time I think we have learned to stain neurons better than Ströbe stained them.

Surgeons who are familiar with the work of Foramitti in using formalinized artery tubes may know that these artery tubes, calves' arteries, were used by Hashimoto in the Russo-Japanese War, and used in the Balkan War extensively. We have used this method in perhaps six or eight cases, but we used with them an auto-transplant. These I have not reported on tonight. In a number of cases we bridged the gap in the ulna of 4, 4½ or 5 cm., by inserting the ends of the resected nerve into the lumen of the formalinized artery. In two of these cases the neuraxes reached the peripheral stump and there was observed regeneration of the peripheral stump, but not so good as when a transplant was used. It is a method that could be used. I feel I cannot state from personal experience that there is not the same amount of connective tissue formation following the use of a formalinized artery as with a fascial tube. The formalinized artery will remain in place and is not absorbed, at least for six months after implantation. The neurons pass down through the lumen of the tube and reach the peripheral nerve stump.

PHILADELPHIA PSYCHIATRIC SOCIETY

Regular Meeting, May 9, 1919

CHARLES S. POTTS, M.D., *President*

DRUG TOXEMIAS, THEIR NATURE, ETIOLOGY AND SYMPTOMATOLOGY. Presented by DR. JOSEPH C. DOANE.

Dr. Doane said that since March 1, 1915, when the Harrison law became effective, much misplaced sympathy had been lavished on the drug taker. Cheap sensationalists had seized on the subject of drug diseases to add to their own incomes, without any knowledge of the subject, or any desire to do good. The stage had also aided to disseminate misconceptions as to the nature of the disease, and such publicity had only added to the number of persons who needed only to know of a vice to become vicious.

The term "drug habit" was not descriptive, for the continuance of the use of a drug was not truly a habit. The physiologic action of the drug did not explain the etiology of drug disease. Symptoms of withdrawal yielded to the drug which brought about withdrawal pain and might partially explain drug abuse. Rather did it seem that drug addiction was but a symptom of some physical, moral or mental abnormality, and that moral and mental degeneracy were but the parents that begot the litter in which drug inebriety, sexual perversion and other social monstrosities were numbered.

Out of a series of 393 cases of drug disease studied at the Philadelphia General Hospital, 267 began the use of drugs on the advice or example of others so afflicted. It appeared, then, that drug disease was but an inevitable expression of some abnormal moral or mental trend, and that this particular form of expression was more or less accidental. Relief of pain, physicians'

prescriptions, ignorance of content of long-continued medication were but predisposing factors that increased the receptivity of an already more or less fertile soil.

Crime and drug addiction were closely linked, for with an increasing need for drug went a decreasing earning power.

Cures were few, temporary relief more frequent, and relapses the rule; but nature frequently cleared the stage of these handicapped persons with her keen weapons—tuberculosis, pneumonia and syphilis.

THE TREATMENT OF NARCOTIC DRUG ADDICTION. Presented by DR. JOSEPH McIVER.

The purpose of Dr. McIver's paper was to relate in a general way the principal factors concerned in the treatment of narcotic drug addiction. His ideas in the treatment of these cases were drawn from observations made on a large number of drug users coming to the Philadelphia General Hospital while he was a resident there.

There were many of the so-called specific methods of treating the drug habitué, but as to the advisability of their use and the permanency of results claimed by some of their promoters, he was somewhat skeptical. With these patients a considerable allowance had to be made for individual differences, and to try to blend them to a machine form of treatment was a very unwise procedure. In the beginning he wished to say that all these cases could best be treated in a hospital or sanatorium. Before any form of treatment was begun, he could not emphasize too strongly the necessity of a complete history and thorough physical examination. A special effort should be made in every case to establish, if possible, the factor that caused him to begin the use of drugs. The treatment of any drug addict was based essentially on the gradual withdrawal of the drug, free purgation and sedatives or stimulants, as might be required in the individual case. For the first twenty-four hours, the patient should be given enough drug to keep him comfortable; by this method, one acquaints himself with just how much of a drug the patient really needs. Patients should be given the same drug they have been accustomed to and by their usual methods. The drug should be withdrawn gradually, and especially is this true with the aged and debilitated. As the drug was being withdrawn, the patients often suffered from nervousness and insomnia; the symptoms could be alleviated to a large degree with very hot baths. The bromids, trional and scopolamin in ordinary dosage were also of great value here. For the nausea and vomiting which were often so persistent, gastric lavage and restriction of diet were perhaps the best remedies. For the pains in the limbs, the so-called withdrawal pains, which were usually very annoying, electric massage proved beneficial. Some of the coal tar derivatives, as aspirin and the salicylates, might be used to advantage. The diet during this course of treatment should be simple and nutritious, consisting principally of milk and eggs.

The one essential factor on which the treatment of the drug addict was based was the presence in the blood of an antidotal toxic substance. That such substances did exist in the blood was proved by Adriano Valenti at the University of Pavia in his experiments on dogs. Taking this as an axiom, the conclusion was easily drawn that elimination was of utmost importance. After the drug had been withdrawn, the patient was usually in a weakened condition.

He should remain in bed on tonics, an especially liberal diet, until he had gained sufficient strength to begin moderate and graduated exercises.

The most difficult stage of the treatment was to give the patient sufficient moral, mental and physical strength to keep him from returning to the use of drugs. His physical strength could be regained by the usual methods employed in physical upbuilding, and of course with this would return a certain amount of nervous and mental energy. After all this was done, however, many of them would still lack the fundamentals of good manhood. A change of environment and good physical condition were necessary for his future welfare.

Conclusion.—1. A careful study of the case before treatment was begun.

2. Free purgation, gradual withdrawal of the drug with stimulants or sedatives, as might be required in the individual case, were the essential features.

3. Any form of treatment that did not consider the presence of an anti-toxic substance in the body long after the drug had been withdrawn and provided for the care of the patient during that time was almost sure to be void of permanent results.

FEDERAL AND STATE LAWS IN RELATION TO THE DRUG HABIT. Presented by DR. JOHN H. W. RHEIN.

The first effort to control the use of opium as a habit by law was in 1729, when Yung Cheng issued an edict against the smoking of opium in China, the penalty being transportation and death. In 1906 again an edict was issued prohibiting the consumption of opium and the cultivation of the poppy. This was the result of the widespread use of opium as a habit in China where 27 per cent. of the males of that country were said to smoke opium. The Chinese nation looked on the habit as a great national, moral and economic menace. In 1909 there was an international conference in Shanghai representing Germany, United States, Japan, China, France, Great Britain, Italy, the Netherlands, Persia, Portugal, Russia and Siam. The object was to regulate the importation and exportation of habit-forming drugs, and to provide for their use medicinally only.

The Harrison act in 1914 was the first law in the United States to be passed for the purpose of regulating the illegal use of narcotic drugs, and the Pennsylvania law passed in 1917 was intended to supplement the federal law, and to strengthen it so as to cover certain features which the federal law, an internal revenue law, did not cover. The recent amendment to the federal law increased the tax and made it unlawful to purchase, sell, dispense, or distribute drugs except in original packages. The primary object of the federal law was to regulate and control the sale of narcotic drugs to the end that they could only get into the hands of ethical doctors and pharmacists.

The effect of the federal law at the present time is to make it extremely dangerous to sell the drug illegally, and the drug vender stands a good chance of going to prison. The federal act had not hitherto been effective so far as the venders were concerned, against whom the accusation was merely possession, unless it was shown that they were vending at the same time. It had not been easy to convict owing to the difficulty in obtaining evidence acceptable to the courts, as the kind of evidence which could convict; recently, however, venders have been brought to time in considerable numbers, and convictions have been accomplished more readily than formerly.

One of the important decisions of the United States Supreme Court was that the law was constitutional which provided that drugs could be disposed

of only on an order written on a form furnished by the commissioner of internal revenue, or on a physician's prescription, and forbidding a physician to prescribe for an addict except in a curative treatment.

The venders apparently have had no difficulty in obtaining the drug. It has often been stolen from wholesale houses. It was believed that there were some of the big dealers in town who were breaking the law, and two or three had already been accused. It has been common to find a prescription for an ounce of morphin at a time and there have been thousands of prescriptions issued for a dram at a time. The state forces have found that the amount of drugs sold by venders is small in comparison with the amount supplied to drug users by physicians. Many physicians who have broken the law technically are inclined to obey the law when their attention is called to their errors in reading or interpreting the law, which was rarely read carefully, if at all.

The drug habit is really a social evil. It flourished mainly with the delinquent; it is as incurable as crime. Removal of the drug from one addicted to the use is a simple matter, but to prevent his returning to it is a social problem, which means the reform of the character of the individual.

The Pennsylvania law is a good one and, if its provisions were strictly enforced, it would practically eliminate the use of narcotic drugs as a habit. But it is one thing to make a law and another to carry it into effect, and to do this would require a personnel many times greater than was provided for and perhaps ever would be. The result is that the drug habit continues to exist and will continue to exist as long as does the criminal class. The drug habit will continue to exist, too, as long as the supply from manufacturers is not restricted. If it were possible to place the distribution of the drugs in the hands of one state official whose business it would be to supply the drugs for legitimate purposes only, the quantity in circulation could be regulated to meet only the need for medical purposes.

DISCUSSION ON DRUG HABIT

DR. F. H. BALDI said that he had had the good fortune of spending six months in France as medical officer with the U. S. Marine Aviation Force, and while there had opportunity to learn of the drug evil in London and Paris. We knew to what extent our own city was infested with the drug evil. It might be consoling, if that were possible, to know that conditions over there were so frightful that they were beyond conception as compared with conditions here.

If the production of narcotic drugs could not be controlled, we would be in a very unfortunate position. All the legislation in the world would not cure the evil. A good plan would be to educate the people as to just what the addiction is, what it means to humanity, and to what it will lead criminally.

Dr. Baldi believed thoroughly in the quick and absolute withdrawal of the drug, and had never seen any deaths caused by it. It was a method which was rather difficult to practice at home, but had worked charms in the Philadelphia County Prison where he had practiced it with most excellent results for over five years.

The cardinal factors in the treatment were: 1. Isolation; 2. absolute control of the patient; 3. deprivation of the drugs; 4. no substitute of any narcotics; 5. elimination by bowels, kidneys and skin; 6. rest, nourishment and work.

CHICAGO NEUROLOGICAL SOCIETY

*Annual Meeting, May 15, 1919*HUGH T. PATRICK, M.D., *President*

SWIFT-ELLIS INTRASPINOUS INJECTIONS FOR GENERAL PARESIS. Presented by DR. PETER BASSOE.

Dr. Bassoe reported Swift-Ellis treatment in general paresis, in twenty-six cases in which the treatment was commenced and in most instances completed during the years 1914, 1915 and 1916.

Of these patients eight are known to be alive; three of them mentally and physically well, three mentally well but physically crippled, one mentally improved, one temporarily improved and now relapsed. Charts were presented showing the laboratory tests and the form of treatment. The largest number of intraspinal injections in any one case was thirty-three. In this patient the first symptoms occurred in 1912, in the form of apoplectiform attacks with transient aphasia. When first seen, in 1914, the patient presented the usual mental and physical signs of early paresis. The treatment has been continued intermittently up to the present time and the patient has been able to be at work almost continuously.¹

Following an intraspinal injection of 32 c.c. of salvarsanized serum in September, 1916, this patient developed a typical picture of "aseptic meningitis," with stiffness of the neck and legs, loss of sphincter control and a cloudy spinal fluid with a cell count of 1,992, mostly polymorphonuclear cells. He recovered from this in a few weeks. The most severe reaction observed was in a man (Case 26) in whom the onset was very sudden in December, 1916, and the laboratory tests were strongly positive. In the course of a week following an intraspinal injection in February, 1917, he developed a spastic paraplegia which has persisted. After six months he was able to walk about the house with the aid of strong mechanical support, and is now slowly improving. Mentally, however, he has been perfectly well since the spring of 1917, and the laboratory tests became negative.²

A man, aged 50, with tabes of long standing, developed mental symptoms so that his case became one of taboparesis. After a prolonged course of treatment his mental symptoms disappeared but he, too, developed a paraplegia of more gradual onset than in the case just referred to. In another case of taboparesis the mental symptoms also cleared up completely and the laboratory tests became negative, but the patient has to walk with crutches on account of a fracture of the neck of the femur which failed to unite. Following the fracture, which was a painless one, there was a brief recurrence of mental disturbance, but during the past two years the patient has been perfectly well mentally and has worked steadily as a clerk in an insurance office.

1. A few weeks after the reading of this paper the patient developed delusions of grandeur and persecution and was committed to the Chicago State Hospital.

2. Detailed histories and charts of this and several of the other cases related will be found in the Chicago number of the Medical Clinics of North America, 1919.

In the three cases with apparent complete arrest of symptoms comparatively little treatment had been given, but in view of the characteristic symptoms and positive laboratory findings, there is some reason to believe that the treatment which was given at an early stage may have been a factor in producing the subsidence of the morbid process.

Dr. Bassoe merely wished to place these case records before the members without making any claims for this mode of treatment. Some of the facts here presented, in his opinion, tended to show that salvarsanized serum is not as inert as is claimed in some quarters, but that at least at times it may produce rather striking changes in the condition of the patient either for better or for worse.

DISCUSSION

DR. RALPH C. HAMILL called attention to the use of mercuric chlorid injections, and stated that for some years he had used $\frac{1}{150}$ to $\frac{1}{100}$ grain of mercuric chlorid dissolved in spinal fluid. The mercuric chlorid was placed in the syringe and then from 15 to 25 c.c. of spinal fluid were drawn into the syringe, allowing the mixture to take place there, and the solution reinjected. There is enough albumin in the spinal fluid to cause a resolution of the albuminate of mercury which is formed by the mercuric chlorid and spinal fluid acting on each other in the syringe. He had succeeded in getting a negative Wassermann by this method when other methods had failed.

As to the meningeal reaction which Dr. Bassoe referred to, in repeated spinal punctures after injections into the spinal canal he had found a very pronounced pleocytosis. He wondered if anything had been discovered as to the curative value of the pleocytes in the sense that they were phagocytic cells and reached the syphilitic foci not reached by the chemical reaction of the medication.

DR. GEORGE W. HALL had been interested in intraspinal work for some time and in the diagnosis of general paresis. He thought Dr. Bassoe would agree that some of us would have to change our criteria of a positive diagnosis if we are to agree with all his diagnoses. In Case 26, in which he obtained such splendid results, he thought it was a rather atypical case of general paresis. In the first place, the patient did not have a positive Lange reaction, and in his opinion, if this reaction is of importance in anything, it is certainly of importance in the diagnosis of general paresis. In his own work, if there was a negative Lange reaction he withheld his diagnosis and allowed the therapeutic tests to aid in the diagnosis. He believed that cases which might clinically be regarded as general paresis, but did not show a positive Lange curve, must be put into the questionable class. He thought a good many of the cases were diffuse syphilitic endarteritis rather than general paresis.

As to the intraspinal medication, he had done a good deal of work in that line at the County Hospital, using the mercuric chlorid and succinimid of mercury, also using it dissolved in the spinal fluid. They were now using serum of the patient's blood, injecting it into the spinal canal and after the injections of the serum administering intravenous medication to see if any results could be obtained in that way. He thought this much less dangerous if results could be obtained. The cell count after injection of the plain serum showed marked increase in the cells, from 2,000 to 3,000 in some cases, and

in others it was not so large. He hoped to be able to report results in the near future. The dose of the mercuric chlorid he thought should not exceed $\frac{1}{60}$ grain but up to that it was perfectly safe. On going higher than $\frac{1}{60}$ grain he had not infrequently had retention of urine and once or twice had to catheterize the patient. If he stuck to $\frac{1}{100}$ or $\frac{1}{60}$ grain, he got no serious results. Caution should also be used when alcoholism complicated the case. He believed spinal medication of any kind was very dangerous in an alcoholic.

MAJOR LEWIS J. POLLOCK said that the futility of ordinary methods of treatment was usually referred to the fact that the choroid plexus was impermeable to drugs. This belief has been based on the work of Goldmann, which has not withstood the test of time. McIntosh and Fildes at first stated that arsphenamin was not neurotropic and in this opinion Ehrlich concurred. Later, when they found that they were unable to obtain from the washings the amount of arsphenamin which had been mixed with a macerated brain, they concluded that the brain tissue did take up some of the arsphenamin and stated that the meninges were impermeable to the drug.

This conclusion was based on an experiment which did not comply with the conditions found in life.

The permeating power of drugs depends on several factors; their passage through the blood vessel, through the cell membrane and the ability of the cell substance to take up the drug. Overton stated that only fat soluble substances would penetrate the brain by an intravital injection. This had not proved to be the case. The permeability of the meninges and vessel walls is responsible for but one part of the success or failure for intravital stains to reach the brain. The permeability of the meninges may be increased by the intraspinal administration of any foreign substance, as was shown by Flexner's work on experimental anterior poliomyelitis. The most important factor is the refusal of the cell contents to take up the drug. This has been proved by the work of Overton, who showed that if the cell membrane be nicked and a gradient formed the substances which would not stain the cell with an intact membrane would not stain it with the membrane punctured. That permeability of vessel walls or meninges is not the important factor was shown by the facts that some crystalloids fail to act as vital stains, whereas colloids do act as vital stains in many instances.

The choroid plexus alone cannot be held responsible for the failure of certain drugs to reach the brain.

DR. WILLIAM ALLEN PUSEY had been much interested in the discussion. He had not treated central nervous system syphilis but some dermatologists had treated a good deal of it and had messed it up, which did not surprise him when experts such as these differed so widely in saying when the disorder was paresis and when meningovascular syphilis. He was much interested in the treatment of late syphilis, and had become rather prejudiced against the treatment of paresis and tabes at present in vogue. The careful reports he had just listened to had not changed his views. He had not been impressed with the conclusiveness of the findings. It reminded him a good deal of the findings in a series of carcinomas in the abdomen and chest treated with roentgen rays, the treatment extending over a few years. Some cases seemed to clear up, but as time went on the hopes diminished.

In Dr. Bassoe's cases the results did not seem conclusive. One patient who received seven or eight intravenous injections and only one intraspinal

injection showed great improvement. Another patient, a physician, received thirty intraspinal injections and a greater number of intravenous injections, and while he seemed better, the intraspinal picture did not improve. In another patient who received forty-two injections, the serologic picture remained unchanged. For a good while he had been seeing some of the untoward results of intraspinal medication and he felt gratified to find that they were not due to poor technic when the same results were reported by experts who were doing the same work.

To sum up, if he had paresis or tabes he would not take intraspinal medication with the hope of getting well, but would let the thing pursue its course as rapidly as it might. To his mind these things were quite analogous to what was being done in syphilis in general and he had given a good deal of thought to what was being done at present. He was by no means sure that the syphilitic was better off today than he was ten years ago. Undoubtedly there were more neuroresidue and renal complications than before. In late syphilis some physicians were trying to make the patients negative in spite of everything, and his notion was that in trying to use the man as a retort they were not helping him. It might help to make him negative, but if he became negative, he came back after a while and in repeating the treatment very often they did considerable damage to the man. He had seen a good many cases of this kind and going back to himself as a criterion, if he had old syphilis he would try to get in good shape clinically and treat his syphilis as well as he could on the clinical data, but he would not undertake to let anyone make him negative by way of the spinal fluid. He thought nothing conclusive had been arrived at.

DR. SIGMUND KRUMHOLZ felt that the cases of syphilis should not be treated intraspinaly. In the advanced cases of general paresis with distinct mental changes, if there were any prospects at all of being cured by the way of intraspinal injections, he thought they should be used. From the reports of Dr. Bassoe, in about one third of the cases he had observed there was improvement. Two or three of the cases went bad in three years, but if they had been ameliorated or almost cured for that length of time it was a great accomplishment, and he thought the treatment should be encouraged. He failed to see why any parietic should be left alone when there was any hope at all from intraspinal treatment. In syphilis of the central nervous system he never used the intraspinal injections, and thought this should not be done because the intravenous injections produced the same results.

DR. SYDNEY KUH said that most of those present knew that before they could make a positive diagnosis of tabes there was a very marked degeneration of nerve fibers in the spinal cord. He thought they all knew that as soon as they could make the diagnosis of paresis there was a degeneration of nerve cells and of nerve fibers in the cortex of the brain. He could see that before there was the least suspicion of any mental change a great deal of nerve tissue was gone. He asked if the gentlemen who reported apparently cured cases after intraspinal treatment believed that the nerves regenerated after this treatment, or any other form of medication, was used.

DR. G. B. HASSIN said that while it was true that in general paresis a large number of cortical ganglion cells are always more or less damaged, many of them may remain intact. In addition, interstitial changes may pre-

vail in the form of miliary gummata, as shown by Strüssler. The miliary gummata, of course, may be responsible for some of the symptoms of general paralysis. Finally, there occur the aberrant types of this disease described by Lissauer and Alzheimer which usually run a more favorable course and give a better prognosis. Probably some of the cases reported here by Neyman and Bassoe as improved belong to this class, especially Bassoe's patient with attacks of aphasia. Such cases usually improve from any method of anti-syphilitic treatment, including the intraspinal one.

DR. CLARENCE A. NEYMANN, referring to the effect of the pleocytosis, said that a few years ago Dr. Sidney Miller had injected a few patients with a very weak solution of sodium nucleinate, and this put the cell count up to 2,000 or 3,000 and caused a true meningitis. In the few cases that were treated in this way good results were not observed, and the treatment was discontinued because of very bad bladder effects. If general paresis were a disease that might otherwise end favorably, he would not advise intraspinal treatment. In a large series of cases without treatment, Cotton had had only 4 per cent. remissions, but in a series with intraspinal treatment there was 25 per cent. of improvement and he therefore thought such cases should be treated. As regarded the intravenous as opposed to the intraspinal treatment, in his worst cases the only death he had had followed prolonged intravenous treatment. This might have been due to the diarsenol brand of arsphenamin; if arsphenamin was pushed beyond a certain point, they were bound to get a bad result and often this trouble was a good deal more serious than the other trouble combined with paraplegia.

Of course nerve tissue once degenerated cannot be replaced, but the nerve tissue that was left could take over the function, to some extent, of that which was lost. He thought it made no difference whether the mercury was introduced into the serum as a solvent or into the cerebrospinal fluid as a solvent.

DR. PETER BASSOE thought the problem of the treatment of general paresis was very much like that of brain tumor. If the brain tumor were allowed to take its course in a series of twenty-six the patients would get worse and worse, go blind and die. If operated on, some would live and some might get well and he felt that in view of the unfavorable prognosis it was justifiable to give treatment that might cause damage in general paresis, just as an operation on the head for brain tumor might cause damage.

In regard to the diagnosis, if the patient in Case 26 did not have general paresis, what did he have? He did not have tabes; he had increased reflexes, unequal pupils and distinct mental confusion and acted like the ordinary case of general paresis. Dr. Hassin brought out a different kind of case which clinically might be called paresis. Some patients had interstitial changes and some parenchymatous changes; the interstitial changes were influenced by treatment. Those cases that came on suddenly had the best chance for improvement, notwithstanding that they had a lot of plasma cells and infiltrations. He did not think it possible to tell beforehand what kind of a case one was dealing with, so it remained a very obscure thing. Many of his patients had very extensive intravenous treatment without improvement, but they showed at least temporary improvement after intraspinal treatment.

MAJOR POLLOCK asked if the patients would have shown the same degree of improvement if the treatment had not been accompanied by the introduction of arsphenamin into the vein to produce the arsphenaminized serum.

DR. BASSOE thought the arsphenaminized serum possibly acted mainly as an irritant.

DR. L. HARRISON METTLER asked if any large number of cases were reported in which intraspinal treatment was used and a record kept in comparison as a control with other cases.

DR. KUH said he had seen institution statistics showing that cases which were injected lived a shorter time than those not injected.

DR. BASSOE said that Cotton had reported a large number of cases treated and untreated; in the treated cases there was 25 per cent. of remissions and in the untreated only 4 per cent. On the whole, institutional statistics were unfavorable. His cases were not of a severe type and would not ordinarily be in institutions.

Book Review

VEGETATIVE NEUROLOGY. By HEINRICH HIGIER, M.D., Warsaw. Authorized translation by WALTER MAX KRAUS, A.M., M.D., New York. Published by the Nervous and Mental Disease Publishing Co., New York and Washington.

A valuable addition to the Monographs on Nervous and Mental Disease is the recent contribution by Dr. Higier. As Dr. Higier observes in his introduction, the vegetative nervous system has received but scant attention from physicians. The translation of "Vegetative Neurology" by Dr. Walter Max Kraus of New York fills a gap left by the textbooks on clinical medicine. Although it is obviously out of the question for the clinician to devote much time to the comparative anatomy and embryology either of the vegetative or the cerebrospinal nervous system, the anatomy and physiology, as well as the pathology and pharmacology of the vegetative nervous system should have fully as much attention given to them as is directed to the anatomy, physiology, etc., of the central nervous system. Dr. Higier's summary of the latest researches in vegetative anatomy, physiology, and pathology demonstrates the importance of involuntary mechanisms of the lower neurons for the maintenance of normal physiologic equilibrium. In the light of vegetative neurology, abnormal function may often be traced to derangement of the sympathetic or of the autonomic systems, or of ductless glands, the secretions of which influence these systems. How much influence such derangements may exert on the development of mental disorders is as yet uncertain, since in the psychoses and psychoneuroses there is set going a vicious cycle involving the cerebral cortex and the spinal and vegetative neurons, the initiation of which is difficult to ascertain. In any event, vegetative pathology appears to be of no little importance to the neurologist and the psychiatrist, as well as to the internist.

After a brief summary of the gross and microscopic anatomy, of the histology and embryology of the vegetative nervous system, there follows a chapter of twenty-eight pages on the physiology of the vegetative system that gives the reader access to the most recent conclusions and opinions on autonomic and sympathetic innervation. Under pharmacology and pharmacodynamics of the vegetative nervous system, the action of drugs (muscarin, pilocarpin, physostigmin, picrotoxin, atropin) and of internal secretions (epinephrin, iodothylin, hypophysin) is discussed in relation to the sympathetic and autonomic systems. The physiologic action of atropin, epinephrin, pilocarpin, and ergotoxin on smooth muscle is summarized by Meyer and Gottlieb's chart, which, however, is not very recent (1911).

The pathology of the vegetative system and the clinical signs and symptoms of vegetative disturbances are discussed in comprehensive fashion. The author supports the theory of the Vienna school that hypertonus of the sympathetic (sympatheticotonia) and of the autonomic (vagetonia) produces distinct types of neuroses capable of clinical differentiation. Loss of that physiologic which results from the reciprocal innervation of smooth muscle is the outcome of overstimulation of the autonemics or of the sympathetics as the case may be. The rôle of the ductless glands in overstimulation of either

the sympathetics or of the autonemics is pointed out; likewise the influence of the vegetative nervous system on metabolism is not overlooked.

The subject of special vegetative pathology is discussed under anatomic headings thus rendering clinical observations on any part of the vegetative system speedily accessible. The entity of the disease vagotonia, a result of the overstimulation of the vagus and pelvic nerves, or perhaps the consequence of a loss of tonus in the sympathetic, appears to be established on a physiologic, as well as on a pharmacodynamic basis. The most satisfactory method of investigating systematically the functions of the vegetative system from the standpoint of pharmacodynamics is presented, together with the most important clinical signs.

Psychologists will find the monograph of interest in relation to the subject of psycho-physical parallelism. The physiologic evidence of viscera-cortical coordination as presented appears to be just as applicable, however, to the monistic theory of psycho-physical unity. James' organic theory of the emotions seems to be supported by the most recent investigations on the vegetative nervous system: in brief, we feel sad because we cry, are afraid because of tachycardia, increased peristalsis, and goose-flesh, etc. Whether this conception of the emotions be accepted or not, the influence of the visceral state, as affected through the agency of the vegetative system, the cerebrum and the mind is well brought out by Dr. Higier. The clinical psychologist, as well as the purely scientific investigator, will find his discussion of Veraguth's psychogalvanic reflex of value, showing as it does the extremely delicate reaction of the sweat-apparatus to psychic stimuli, a reaction much more sensitive than the pupillary reflex.

Under the heading "Endocrinous or Interrenal Secretory Glands" a brief summary of the physiologic and pathologic functions of the internal secretions is presented, a summary of eight pages giving the results of the latest investigations on this subject—one that is of so much theoretic and practical importance at the present time.

The contribution is unfortunately lacking in adequate diagrams. The inclusion of a diagram of the vegetative system, such as that of Göttlieb and Meyer, would have served as an excellent visual summary of the facts presented in the chapters on physiology, pathology and pharmacology. The three diagrams appearing in the work are instructive, if limited in scope. One portrays the course of the autonomic and sympathetic fibers in the cranial nerves; one, the innervation of the pelvic viscera; and one, the innervation of the male genito-urinary apparatus. These are illuminating, but do not present a unified picture of the whole vegetative complex, a desirable feature of so intricate a subject.

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SOME NEUROLOGICAL ASPECTS OF RECONSTRUCTION *

HARVEY CUSHING, M.D.

BOSTON

A few weeks ago I received a visit from two recently discharged soldiers who may be designated as Pte. L. and Pte. A. The former, a cripple, was being assisted by a young man still in uniform, to all appearances in perfect health. I shall briefly tell their separate stories.

Pte. L—, a member of the organization with which I went overseas, had been the victim of an air raid on Sept. 4, 1917, when several bombs were dropped on our hospital compound, resulting in many casualties. The reception tent, in which he with several others was on duty, received a direct hit. Two were killed outright, and he was so seriously wounded that a double amputation—the right thigh and left lower leg—was necessitated. A few hours later, owing to a galloping gas infection due to the multitude of indriven splinters from the badly contaminated wooden floor of the tent, the left leg was again amputated this time in the mid thigh by (then) Capt. E. C. Cutler.

Pte. L— recovered. In due course he was evacuated to a base port, and after the customary delays was returned home where he received the abundant sympathy, care and favor apportioned to amputés. After being fitted with artificial limbs on which he is learning to walk with some assistance, he finally received his discharge on Feb. 10, 1919, with a surgeon's certificate of 100 per cent. disability entitling him with his insurance to a compensation of \$157.50 a month. An intelligent and ambitious young man, he is now a student at the Institute of Technology.

Pte. A—, the soldier who accompanied and assisted the cripple, was a fine appearing young fellow over whose prospects, however, his friend and companion expressed great concern. A private in the 26th Division, he had received a head wound, with immediate loss of consciousness, Sept. 12, 1918, in the early hours of the St. Mihiel attack. He appears to have been removed promptly from the field, to have passed the 101st Field Ambulance, and to have reached Mobile Hospital No. 1, stationed near Ancémont, some 10 miles from the line. There he was operated on about twelve hours after the injury by (then) Capt. Charles E. Dowman, the senior officer of one of the neurosurgical teams. As the members of these teams kept and forwarded to me duplicate notes of their head cases, Capt. Dowman's record of this man's operative history was in my possession. To illustrate the character of the observations

* Read before the Congress of American Physicians and Surgeons, June 16, 1919, at a symposium on the subject of Medical and Surgical Reconstruction.

made by the officers of these hurriedly assembled teams, to all of whom I am under lasting obligations, this record may be given in full.

A—, John C., No. —, Pvt. Hqs. Co., 102 F. A. Left parieto-occipital gutter wound with dural and brain penetration of bone fragments.

Admitted Mobile Hospital No. 1, 11:10 a. m., Sept. 12, 1918. Tagged 101 Amb. D. S., 9 a. m., Sept. 12, 1918.

A. T. S.: Yes. A history of the exact time of injury or how patient had been injured could not be obtained on account of an apparent aphasia. "No signs of paralysis; vomiting at 10 a. m." noted on diagnosis tag.

General Condition:—Patient conscious but unable to talk. Pulse 54; blood pressure 130-75-55.

Wound:—Transverse, tangential, left occipital region, measures 10 by 4 cm. ragged edges of torn scalp; exposed brain with evident loss of brain substance. Roentgen-ray report: "No foreign body in brain. Indriven fragments of bone, left occipital region. Foreign body (high explosive) 18 by 10 mm. in lower end of right tibia."

Neurological Findings:—Aphasia. When asked when he was wounded answered "tonith." Recognizes objects but is unable to call their names properly. Calls a match "penth," and later calls it "menth." Agraphia. Unable to write name, though he spelled his name J-O-H-N as he attempted to write it. Loss of muscle sense in right hand. (Is right-handed.) No loss of sensation to pin-prick right or left. Slight weakness (emotional) right side of face. Right hand grip weaker than left. Pupils dilated, but equal right and left. Abdominal, epigastric and cremasteric reflexes absent right and left. K. K. hyperactive both sides, more so left. Achilles tendon active right and left. Babinski reflex present, right, suggestive left. A complete right homonymous hemianopsia.

Operation:—Sept. 12, 1918, 8 p. m. (about 12 hours). Pulse had come up to 80 per min. Novocain-epinephrin after preliminary one-third grain morphin. Three-legged incision made with excision of wound in scalp; block removal of bone; débridement of brain by suction, with removal of numerous bone fragments and considerable contused brain substance. Wound in dura was 2 by 1.5 cm.; treated by excising soiled edges. Two bleeding points tied with fine silk. Scalp closed with interrupted silkworm gut. A small foreign body removed from scalp of vertex. No drainage.

Gunshot wound posterior surface right leg, 5 cm. above ankle. Under novocain wound was debrided and shell fragment removed from lower end of tibia.

Postoperative Course:—Sept. 13. Pulse 100; patient brighter. Tries to talk but is unable to call objects by their proper names. Eye grounds normal.

Sept. 14. Pulse 80. Able to say a few words. Strength in right hand improved; general condition good.

Sept. 15. Every other stitch removed. Healing reactionless. Aphasia improving slightly. Brain pulsating.

Sept. 17. All stitches removed. One or two drops of cloudy fluid from center of wound. Aphasia improving. Right hemianopsia the same.

Sept. 21. Evacuated.

The subsequent story as I learned from my two visitors was as follows: From Mobile Hospital No. 1 he was removed to an evacuation hospital stationed near by; then after two weeks as a stretcher case to Base Hospital No. 26 at Allerey, then to St. Nazaire and finally to Brest. By this time he was able to be up and about and on Nov. 11 he was returned on an army

transport. He landed at Newport News and was sent from there to a base hospital, from which place, being so far recovered that he supposedly could travel alone, he was discharged December 7 with orders to "report for duty" at the convalescent center near his home. He was found wandering about the once familiar streets of Boston, of the names and direction of which he had lost all recollection; an ambulance was secured and he was taken to the local army hospital where arrangements were made for his admission. He remained there four months.

Before his enlistment he had been an expert accountant; and as he had completely lost his ability to deal with figures he was assigned to a class in the hospital with others who were learning arithmetic. The exercises, however, according to his statement, were so far beyond him that he merely became confused and soon ceased to attend them. Finally on April 7, 1919—"condition: maximum improvement obtained"—he was brought before an S. C. D. Board of junior officers (neuropsychiatry not represented) and was given 90 per cent. disability. This entitled him to a compensation of only \$27.00 a month, for curiously enough any percentage disability below 100 per cent. is reckoned on a \$30.00 a month basis.

I shall resist the temptation to go into the details of the young man's residual symptoms further than to say that mild excitement, stooping, or any slight physical exertion, causes his brain to "pound" as he describes it. There are occasional periods of numbness and contraction in the right hand and arm suggestive of early jacksonian seizures. Though his hemianopsia has subsided, there remains a marked sensory aphasia (agraphia), lacunar amnesia and a definite slowing of his mental processes.

I am aware that these circumstances may be unusual; that the status of both these men is subject to revision; that judgment even by an expert, if one is to be had, regarding the future of post-traumatic mental derangements is difficult and often fallacious. I fear, however, that in this recital of a chance episode I am merely telling what in the main can be duplicated in most, if not all of the many hospitals which receive the victims of these craniocerebral injuries.

The story has been told, however, in order to compare the treatment and the reward afforded to a soldier without legs who has resumed his studies and will be perfectly capable in all probability of earning his livelihood, and the soldier to outward appearances physically intact, who has a cerebral defect with a residual abyss in his mental processes which no amount of reeducation will ever enable him completely to bridge and who suffers therefore from a handicap incomparably greater than the amputé, so far as his future outlook is concerned. Both Pte. L. and Pte. A. know this as well as I.

Now the subject which has been apportioned to me for this occasion is the "Neurological Aspects of Reconstruction," and for several reasons I suffer from considerable embarrassment in approaching it. In the first place the word "Reconstruction," useful as it may have been and still is as a means of focusing public attention on an idea —

like the word "Conservation"—appears to me to be in danger of being so greatly overworked that the medical profession is likely to forget that it merely represents in our present connection the later treatment—the making over—of our disabled soldiers. It is precisely what, admittedly in smaller scale, the personnel of the medical and surgical and social service departments of every well-conducted civil hospital has in the past attempted to do for its patients—to restore them to a life of greatest usefulness. Indeed, in some communities, like my own, there is what is called a Tide-Over League where occupational therapy, possibly begun in the wards, is continued; and this is supplemented by the State Industrial Accident Board which has a department for vocational training. The process in each case is the same, and I have some misgivings, lest lulled by the magic of this shibboleth, we may fail to observe that the same defects in our existing hospital organization, in our professional training, and the same unfamiliarity with the more difficult bypaths in medicine, particularly those relating to the psychology and behavior of the individual, exist as before.¹

Another source of embarrassment lies in the fact that my particular relations to the wounded soldiers have been largely confined to the more acute aspects of treatment—the immediate saving of life by the early operative intervention in the case of craniocerebral wounds. Regarding this early stage, alone, of the process of making over our wounded, in so far as it has been possible overseas to influence it from a neurological point of view, I can speak with some familiarity, and what I may have to say of the subsequent stages of this and other neurological problems represents the impression merely of an interested and sympathetic onlooker.

Unfortunately, as physicians and surgeons, we in general know less about the nervous system and its disorders than we do of the disorders of any other part of the body. Despite its unquestioned importance from a broad national and sociological point of view, as well as from

1. In many civil hospitals, indeed, efforts have been made to introduce elementary vocational training but, as seems to be the case with the majority of our wounded soldiers, patients are loath to go to school in a hospital. In the army hospitals I have visited, the shops and classrooms, alas, tend to be empty, and particularly those in the larger cities have, in the shape of an entertainment committee, a serious competitor for the soldiers' favor. In some institutions this has reached such a stage that it has been necessary to issue orders for convalescents to go into the shops for a certain number of hours a day on the basis that the work was an essential part of the treatment of their disability, as it often is. In the long run the men who are privileged to wear stripes make admirable patients as long as there is anything serious the matter with them, but subsequently they become difficult to handle. As one of them put it to me "they don't see the use of spending their time in shops making toys for children."

the standpoint of each person, the subject, possibly due to its complexity, is largely shunned, and the mental hygienists, the psychiatrists, neurologists, and more recently the neurosurgeons of the country who are endeavoring to be heard but represent a small voice, and that not a concerted one, to which Medicine in general has paid scant heed, and which is now quite lost in the uproar of Reconstruction.

Unquestionably, the wear and tear of modern life falls most heavily on the nervous system. This is true of peace no less than war, but in time of war with its additional strain, shared by all both in and out of uniform, sane minds are in the long run more essential to the country's welfare than sound bodies, important as these are. I recall with interest the nature of the official physical examination to which, in May of 1917, the volunteers enlisting for our hospital unit were subjected. It was a matter largely of teeth, of toes, of stature and of inguinal rings. In the absence of mental or neurophysical tests many candidates who would have received high marks on these scores were rejected, and the reverse was equally true. Fortunately, after the draft was adopted the character of these examinations was greatly improved. Psychiatrists appeared on the examining boards and a large number of prospective soldiers showing neuropathic tendencies, who obviously would not endure the rigors and restrictions of military training, were screened out together with the physically unfit. The startling thing from the standpoint of our national well being was the number of cases so rejected.

In spite of this careful combing out, a large number of the accepted, when subjected to the strain and disciplinary restraints even of camp life near the theater of war, developed psychoneuroses which proved their unfitness for service; and, as you well know, a great many soldiers with nervous systems sufficiently stable to withstand these lesser trials broke down when the influence of high explosive was added to the stress already imposed by fatigue and excitement.

No compliment too great can be paid to the organization with its series of hospitals which Colonel Salmon established in France to meet these problems, and the recent report by Lieut.-Col. Sidney I. Schwab² of what he calls the "A. E. F. conception of the war neuroses" is one of the notable contributions to psychiatry which has come out of the war.

To these aspects of the disorders of the nervous system the entire energies of the neuropsychiatrists who were in service overseas was concentrated, as it is perhaps essential that they should have been, in

2. The War Neuroses as Physiologic Conservations, Arch. Neurol. & Psychiat. 1:579-635 (May) 1919.

view of the incessant wastage which was known to have occurred in the allied armies from these sources. The problem was urgent; the treatment to be effective had to be put in operation before opportunity arose for the neuroses to become a fixed habit.

As the few neurologists who were available became engulfed with the psychiatrists in this important work, the nervous disorders of organic origin were perforce somewhat neglected, though it was apparent to all that these cases would sediment in our hospitals and represent the final precipitate of the war injuries — and not only the last, but the most difficult with which to deal.

Meanwhile, under a program of which we had scant tidings abroad, military surgery in our army was variously subdivided into general, orthopedic, genito-urinary, and so on, with a department for surgery of the head to include the diseases and injuries of the eye, ear, face and jaws, as well as of the brain itself. Though this was a sufficiently workable program, it unfortunately did not fit in with the plans adopted overseas, and the decision was made, I believe, in the spring of 1918 to place the injuries of the nervous system as a whole under a department of neurological surgery. This would seem the more natural arrangement, and it appears from the syllabus of the courses³ offered in the excellent neurosurgical schools which came to be established in New York, Philadelphia and St. Louis, where the instruction was in the nature of a general survey of the peripheral as well as the central nervous system, that the final tendency at home was in the same direction.

It was not until June, 1918, that a neurosurgical consultant was appointed for the A. E. F. and authorized to organize a service. No precedent covering the activities of such a department existed either in the French or British armies, nor were there any available figures that would serve to give an idea of its probable responsibilities beyond the rough computation from a series of 10,000 wounded in the French 6th Army that 25 per cent. of all battle casualties presented neurological problems of one sort or another; that the major peripheral nerves were

3. These admirable courses in which neurologists and neurosurgeons combined were such as might well be continued as graduate courses on a peace basis for which they were perhaps better adapted than for the actual surgical treatment of war wounds which, as a matter of experience, can only be learned where they occur. These schools, however, have well justified their establishment if they did nothing more than increase the general interest in neurological surgery, and doubtless with the less urgent though equally important reparative neurological work which is still to be done, the labor which the instructors put into these courses will justify itself in the broader neurosurgical point of view of their many pupils.

involved in 20 per cent. of all serious injuries of the extremities, and that wounds of the head, including all types, represented 16 per cent. of all battle wounds.

Estimates such as these based on figures secured from certain engagements in certain periods of the war may, however, fail to represent the percentages which occur under changed conditions of warfare. In the open battles in which our forces came to participate, with almost as much actual exposure to machine-gun fire as to high explosives, the injuries on the whole were altogether of a different character, far less severe and far less provocative of sepsis than those to which some of us had been accustomed during the preceding year as a result of the battles in Flanders. For although the enemy's system of machine-gun defense by isolated concrete emplacements was first encountered during the Passchendaele operations, high explosive was nevertheless so predominant as to be the cause, at least in the case of head wounds, of practically all the examples.

It was the custom in the British army to cluster in twos or threes their casualty clearing stations (comparable to our evacuation hospitals) which had been developed during the war. During the tragic months of the battles for the Ridges, one of these hospital clusters behind the 5th Army was given over entirely to the reception of the army head cases, together with the walking wounded and the gassed cases from the nearest corps. By this arrangement a sufficient number of beds was made available for the retention of craniocerebral injuries which, as experience has shown, do not stand transportation well after operation, but must be held like other wounds in which primary closure has been attempted. Though the pressure of work was great at times,⁴ the conditions were nevertheless ideal for the encouragement of detailed clinical observation. Whenever opportunity offered, there were inter-hospital meetings; accurate though brief clinical records were taken on duplicating books which were provided, and through the agency of postcards supplied by the British Research Committee—cards which accompanied the patient on his evacuation and carried the surgeon's address—a follow-up system was possible so that prompt returns could be secured from both base and home hospitals, and a final report of the condition, if requested, was made by the Research Committee on the patient's discharge from service or resumption of duty.

4. For example, during the first two weeks' period, July 26 to August 8, including the single opening battle of July 31, there were 1,017 head cases admitted out of 9,103 admissions.

IMPRACTICABILITY OF BRITISH FOLLOW-UP SYSTEM FOR
AMERICAN ARMY

So far as circumstances permitted, the effort was made to introduce the main features of this system for the benefit of the neurosurgeons operating in advanced hospitals in our own army.

The essential functions of an army hospital are the registration of the wounded and their evacuation. Treatment, aside from first aid, necessarily comes last, and the farther forward the hospital is situated the more closely must this order of obligations be observed. Nevertheless, an army hospital, no less than civil hospital, has unwritten obligations which are often neglected and which lie apart from its administrative work—the detailed care of the individual patients, the training of junior officers representing the students, the proper utilization of material for the furtherance of our knowledge of the problems confronting us. Admittedly, research does not often thrive within the sound of battle, but nevertheless a spirit of inquiry may be kept alive. For such a low order of investigation as the mere study and tabulation of groups of cases and their end results is not infrequently of very great value.

The conditions in our overseas army medical units were very much like those of the divisional units. Medical officers, like battalions, often had to be thrown into gaps regardless of special training or fitness, and it is perhaps astonishing that they accomplished as much as they did. There were, doubtless, many misfits—men in the line who had never fired a rifle—surgeons in forward hospitals who had never seen a war wound and who had been instructed at home in such principles as the Carrel-Dakin treatment or the primary closure of wounds, neither of which could be put into practice under the conditions which for the most part confronted us in the A. E. F.

That good work may be done and careful records kept under these circumstances is exemplified by the clinical note from an evacuation hospital which chance had led me to quote, but I must confess to the fact that with us a follow-up system was impracticable; the records for the most part were poorly kept or not at all, for few organizations had any field medical cards; and that even when—as was true of the duplicates of the notes I have quoted—they *were* sent on with the patient they rarely reached their destination.⁵ Surgeons were thus precluded, in forward areas at least, from any possibility of acquiring personal information, much less of advancing general knowledge, by

5. In the hospitals in which I have made inquiry I am told that about 75 per cent. of our wounded arrive at the base hospitals here with proper army forms, but very few of them with any clinical records whatsoever from overseas.

the study of end-results except by hearsay evidence of the condition in which their operated cases reached the base. Moreover, by the time the July offensive began, things moved so rapidly with our entire army organization that little more could be done than to locate as many surgeons as possible who had had neurological training or even interests, to equip them so far as could be done with proper tools and see that each mobile and evacuation hospital had at least one neurosurgical team reasonably capable of properly caring for injuries of the brain.⁶

Organization and Work of American Neurosurgical Teams.—The story of these neurosurgical teams, as well as of the general situation in France, is told in a final report to the chief surgeon. This will be found in full in the author's reprints.

We are at the present juncture much more concerned with the problems now confronting us than with those of the past. During our last weeks in France, representatives of Colonel Salmon's department and of my own had been stationed at the chief hospitals at the ports of debarkation, with the object not only of tabulating all of the cranial, spinal and peripheral nerve cases, but of insuring the fact that they did not leave France unaccompanied by a detailed note of their neurological condition. We expected that at the port of entry a sorting station would be established, and that the from 3,000 to 4,000 neurological patients would be routed thence to one or two large centers where would be stationed medical officers particularly competent to handle them—psychiatrists, neurologists, neurosurgeons and orthopedists, selected on the double score of greatest experience in dealing with these particular conditions and greatest likelihood of advancing our incomplete knowledge by a thorough comparative study of this large material. Such an establishment, coupled with a laboratory for experimental neurology which Major L. H. Weed had so profitably conducted in Baltimore during the war and profiting by the studies made by Professor Huber at Ann Arbor, would have put our contributions on a parity with the brilliant studies which have been made during the past four years by the British and French neurologists, and carried over to civil life might leave us with a distinctly American "School of Neurology" under a permanent and central organization.

Similar ideas were in the minds of those at home, but something more than the ocean was interposed between our overseas and our

6. The importance of special training for this kind of work may be gathered by quoting the figures from one hospital during a certain engagement. In a series of thirty-eight cases of dural penetration operated on by the single neurological team, there was an operative mortality of 29.4 per cent., whereas twenty-six cases operated on by eleven different surgeons without equipment or training, in the same hospital, gave 62 per cent. mortality.

home programs in this and I fear in other departments as well, which not even wireless nor the cable seemed able to penetrate.

It is perhaps understandable why the more natural and effective plan was not adopted at the outset. There were many influences at work and the result has been that neurological patients, like most others, have come to be more or less widely scattered in many army hospitals, and short-sighted parents have even gone so far as to remove their wounded boys from army auspices in order to send them to their own choice of physician. But he is therapeutically helpless when compared to a group of medical officers who have numberless examples of the same lesion under their observation, with every facility at hand for the elaborate studies and after-care essential to the best results of surgery in this difficult type of case. This work has been done admirably at some hospitals like those at Cape May and Fort Sheridan, but we cannot reduplicate superior ability indefinitely—the special cases must be routed to the place where ability is available and this, I think, we are going to find true when we come to translate the lessons of military to industrial surgery. Unquestionably, a program of reconstructive treatment for disabled soldiers in a modern hospital is much more likely to be effective than any similar program for the care of industrial injuries, unless done in the mass and on a state or national scale, because the treatment and reeducation can be enforced on reluctant and short-sighted individuals, because inter-departmental cooperation can also be enforced, because the number of cases of a given type can be brought together so that wide experience with a given problem can be acquired, and lastly, because in the army, medical officers are giving—or should be able to give—their full time to these problems, undistracted by administrative details or by outside calls on their time.

We are tending to fall short of our immediate opportunities for a number of reasons, some of which I have mentioned—the insistence that the wounded be distributed to hospitals sufficiently near their homes so that relatives may have access to them; the irresistible tendency on the part of parents to remove wounded soldiers from army auspices and place them under the care of their local civilian physicians or surgeons; the desire of medical officers to withdraw from the service; the unquestioned let-down in the activities of all war-weary people which followed on the armistice and which seriously militates against the effective carrying out of policies already accepted and seriously blocks the adoption of any newly constructive ones.

So far as the neurological cases are concerned, though late it is not too late to reassemble them, for as I have indicated they will sediment in our several hospitals as the dregs of the casualty lists when

nearly all others have been discharged. It is understood that steps are now being taken in this direction and it is wise, for neurological problems are very special ones, whether they lie in the province of the neurosurgeon or the psychiatrist, and the proper sort of operation, if any, no less than the proper sort of vocational training to be given to a soldier with a cranial defect and perhaps subject to epilepsy, may well tax the judgment of experts far more than other problems of surgical reconstruction.

We hope, therefore, that the desired congregation of these cases may be brought about and possibly under the auspices of the War Risk Insurance Board and the Department of Public Health a group of men may be gathered, capable of carrying out at least a portion of the program which some of us have so ardently longed to see established, not only for the immediate benefit of the soldier, but also for the future of neurology through our increased knowledge of the disorders of the nervous system. When that time comes there will be less occasion for men with an old craniocerebral injury to wander, like Pte. A., into a doctor's study and ask if their country has treated them as fairly and understandingly as the men who lost their limbs in its service.

It is but human that the country shall tire of its wounded as a weary and harassed physician comes to tire of his patients, and as my hearers may doubtless become wearied of this abundant talk of Reconstruction. But what we, as physicians and former medical officers, must all set our faces toward is the greatest possible good which the country may derive from the lessons and experiences and indeed the mistakes of our corps during and after the war.

One very essential transfer of experience can take place between the treatment of war wounds and the treatment of industrial injuries which annually far outnumber our battle casualties. This has come to the minds of all, and mayhap a new profession for women, as important as the nursing profession, may arise through their valuable services as reconstruction aids.

We can all foresee many difficulties, even greater difficulties than confront us with our present problem, for victims of these injuries will be still more widely scattered among hospitals than our battle casualties have been. But this fact need not discourage us, particularly since, with all this demonstration of what may be done to rehabilitate our sick and wounded soldiers, the people and the nation are aroused, and private and governmental agencies will be more ready than ever before to aid the profession in its efforts to extend its activities—indeed will expect the profession to extend its activities beyond the hospital wards and operating room into a department of Civil

Reestablishment, if I may use the term of our Canadian friends for Reconstruction.

The medicine of our immediate forebears largely concerned the care of the individual when subject to injury or disease. Fine as the record may have been, we are going to see it eclipsed in the present century by two far greater movements that come before and after this period of hospital treatment. One of them is already well under way—preventive medicine, and the public health service; the other, the civil reestablishment on a large scale of the nation's sick and injured whereby they may be returned as part time participants at least in our industrial army. When medicine assumes this latter function on a large scale, we may properly come to attribute it to the example set by the Medical Corps of the Allied and our own armies to give the wounded soldiers of the past war their just dues.

CUTANEOUS SENSIBILITY IN CASES OF PERIPHERAL NERVE INJURY: EPICRITIC AND PROTOPATHIC HYPOTHESIS OF HEAD UNTENABLE

STANLEY COBB, M.D.

BOSTON

INTRODUCTION

Lack of standardization in the methods of examining sensibility in cases of peripheral nerve injury has led to diversity of results, and thereby to differences of opinion regarding the physiology of the peripheral nerves. Neurologists in different clinics often use totally different methods, and two men in the same hospital often get incompatible results in their sensory examinations of the same patient, because of apparently minor differences of technic. Another source of confusion lies in the terminology used to describe areas of anesthesia. A large part of this report is therefore devoted to reviewing the literature, in an attempt to gather from recent physiologic investigations facts applicable to the clinic. Too many workers have been willing to accept textbook interpretations of Head's work without reading the original papers themselves. Thus the popular conception of Head's theory has become more simplified, more attractive and farther from the facts, while the work of Boring, Trotter and Davies has received little attention.

REVIEW OF LITERATURE

The experimental investigation of cutaneous sensation began with the work of Head and his collaborators,¹ 1905-1908. Previous to this time, the skin had been studied merely by the introspective method, but the careful work of Goldscheider and Von Frey had discovered the main facts of the punctate distribution of end organs for each of the principal modalities—touch, pain, heat and cold. Barker² was the first to study an area of anesthesia by these methods, and his findings in an area of anesthesia on his own arm caused by a cervical rib, corroborated the physiologic findings of von Frey.

By having the sensory branch of his own radial nerve divided, Head brought out strikingly the fact that pressure was a subcutaneous sensation, and that light touch had to be very light in order not to elicit

* From the surgical service of Lieut.-Col. Charles H. Frazier, U. S. Army General Hospital No. 11.

1. Head and Rivers: *Brain* 31:323, 1908.
2. Barker: *J. Exper. M.* 1:348, 1896.

this underlying deep pressure. He carefully followed the return of sensation to his area of anesthesia for nearly two years, and from the data thus accumulated he evolved the theory of "epicritic" and "protopathic" sensibility.

"Epicritic" includes: (a) Recognition of light touch, as with cotton wool; (b) thermal sensations between 25 and 40 C.; (c) localization of cutaneous impressions; (d) discrimination of two points (compass test).

"Protopathic" includes: (a) Cutaneous pain of all kinds; (b) heat above 45 C.; (c) cold below 20 C.; (d) mechanical stimuli to hairs.

Head's general theory is well summed up by Boring³ in these two paragraphs:

Cutaneous sensibility is mediated by two afferent nervous systems which, from an evolutionary standpoint, are of different age. The older and more fundamental system is the protopathic; the later system, which represents a higher development, is the epicritic. Ordinarily, in most cutaneous experiences, the two systems act together. Under certain conditions, however, notably those following nerve-division, there may result a dissociation: after the division of a nerve the loss of "epicritic" sensibility is generally more widespread than the loss of "protopathic," so that there results a region supplied by protopathic sensation alone; but the relative distribution is not always of this kind, for there may occasionally appear areas which are supplied by "epicritic" sensibility alone. There is usually also a temporal dissociation: "protopathic" sensibility returns after nerve section before "epicritic," which usually does not begin to return until the return of "protopathic" is almost, or entirely, complete. Under normal conditions there is always a functional dissociation; for the internal organs and some other regions of the body, such as the glans penis, are supplied with "protopathic" but not with "epicritic" sensibility.

In the normal skin the two systems function together. Not only do they supplement one another, but there is also an inhibitory effect of the "epicritic" on the "protopathic" system. The latter naturally mediates intense sensations which are badly localized. The addition of the "epicritic," however, inhibits the bad localization of the "protopathic" and partially inhibits the intensity, besides adding its own complements to sensation.

This theory was accepted and taken up by the textbooks, gaining a hold that has scarcely been shaken by the more recent work, which has not only failed to corroborate it, but has pointed out the fallacy of the whole theory.

In 1909 and 1913, Trotter and Davies⁴ published papers describing repetitions of Head's work. Their experiments were more extensive, seven different nerves being divided and sutured in one or the other

3. Boring: *Quart. J. Exper. Physiol.* **10**:1, 1916.

4. Trotter and Davies: *J. Physiol.* **38**:134, 1909; *J. Psychiat. u. Neurol.* **20**:102, 1913.

of the authors. Their methods of examination were also better than Head's, since they were from the beginning able to benefit by his experience, and their critical analysis and repetition of his work seems fair and well founded. In general, they state that all forms of sensibility tend to reappear together after nerve division and suture, and that all returning sensation is at first hypoaesthetic, gradually approaching normal sensitivity.

In the text of the paper, the statements are elaborate and direct. The idea that there are separate fiber systems for moderate (epi-critic) and for extreme (protopathic) perception was not borne out by the experimental facts, because when recognition of moderate degrees of temperature was lost, the extreme degrees were felt as moderate. That is to say, there was a thermal hypoaesthesia, but not a loss of one or two hypothetical forms of sensibility to temperatures. Again in areas of tactile hypoaesthesia the ability to discriminate two points was found diminished but not lost. So, too, with pain; it was found to return quantitatively, for at the very center of an hypoaesthetic area, a heavier prick was necessary to cause pain than at the periphery. Finally, they state that Head's areas of dissociation merely represent varying grades of hypoaesthesia, and that they doubt more and more the capacity of Head's hypothesis to generalize the facts.

In 1916 Boring³ published a monograph summarizing all previous work and reporting his findings in an area of anesthesia obtained on his own arm by cutting and suturing the anterior branch of the internal cutaneous nerve. Whereas the researches of Trotter and Davies were more extensive than Head's, Boring's were more intensive. He was a trained psychologist, and for thirteen months before the operation, he trained himself to recognize quantitative and qualitative values over the area that was to become anesthetic. Then after the operation most careful observations were made, with both qualitative and quantitative readings, for a period of over two years.

The results are clean cut, and in essentials corroborate and amplify the findings of Trotter and Davies. In general the abnormalities of all four modes of sensibility occur in the same region, but as regards the specific spots, there is no coincidence at all. Return of sensation is found to be gradual, from anesthesia through hypoaesthesia to normal. It is not easy to state for a gradual recovery what the order of return of the modalities may be, but curves of the quantitative readings were made and it was found that pain appears to return to a normal state earlier, only because its return is more abrupt. In reality it reaches normality at about the same time as cold sense and touch (called "cutaneous pressure" by this author). The sensibility to warmth seems to lag behind somewhat.

In his criticisms of Head's hypothesis, Boring states that it stands in peculiar isolation with regard to the work of other investigators, that the evolutionary grounds for it are unique and invalid, and that the results of his own experiment do not bear it out. Neither he nor Trotter and Davies, found the dissociation of areas of epicritic and protopathic sensibility.

Thus careful experimental work is found to refute Head's hypothesis. A review of the clinical literature on peripheral nerve lesions will now demonstrate that it has been found unsatisfactory in that field also.

Dejerine and Mouzon⁵ found that in general the areas of anesthesia to brush or cotton tampon corresponded to those for pin prick. They therefore usually tested with prick alone, but occasionally amplified the examination with heat, cold, and vibration tests.

Tinel⁶ in his book on nerve wounds says:

Tactile, painful, and thermal sensibility should be studied in succession.

In reality, this minute examination is not usually necessary, for the areas of the three sensibilities are usually almost identical. It may at the same time be stated that thermal anesthesia is a little more widely diffused than painful anesthesia and the latter than tactile anesthesia.

But here again we are liable to an error of interpretation, for in the case of each sensibility we must distinguish the coarse sensation from the fine appreciation of the qualities of the sensation. This is the distinction, set up by Head between protopathic and epicritic sensibility; the vague sensation of touch is to be distinguished from the clear appreciation of the nature of the contact and of its precise localization; the rudimentary sensation of pain must be differentiated from the ability to distinguish the quality of the pain; the differentiation between hot and cold must be distinguished from an exact appreciation of moderate temperatures. There are so many special sensibilities, corresponding to terminal apparatuses, all the more complex because they supply more precise notions; in nerve sections they disappear with a rapidity proportional to their complexity and become regenerated all the more slowly as they correspond to apparatuses more highly differentiated.

Practically, in the case of peripheral nerves, we may generally dispense with these minute examinations.

Exploration with a pin alone supplies all necessary information.

The main fact gleaned from this quotation is certainly that in peripheral nerve lesions the areas of anesthesia for the various modes of cutaneous sense practically correspond, so that one test suffices for all. This is a practical refutation of Head's hypothesis, yet the author seems to accept the theory. His review of it is inaccurate in an essential point, for he says: "In the case of *each* sensibility we

5. Dejerine and Mouzon: Presse méd. 30: *ibid.* 31.

6. Tinel: Nerve Wounds, New York, William Wood & Co., 1917.

must distinguish the coarse sensation from the fine appreciation of the qualities of the sensation. This is the distinction set up by Head between protopathic and epicritic sensibility." A careful reading of Head's original paper shows that of the fundamental types of cutaneous sensation, one is classed as purely "epicritic" (light touch), one as purely "protopathic" (pain), and only the thermal senses have adaptations for appreciating both coarse sensation and the finer qualities. Mechanical stimulation of hairs might be considered the protopathic analogue of light touch. Localization and two-point discrimination would seem to result from the arrangement of tactile end-organs and are therefore not modalities of sensation. Thus it cannot be derived even from Head's papers that for each sense there are the two types of innervation, although his argument and discussion strongly suggest this conclusion. Indeed, as Trotter and Davies⁷ say, "Symmetry and the desire for classifications are apt to be mistaken for physiological principles."

The English clinicians are naturally even more prone to use the old classification. Souttar⁸ uses a camel's hair brush for "epicritic," and a pin for "protopathic," but states that in general the difference between the areas is negligible (p. 281). He also says that "deep sensibility may be elicited by moving the hairs whose roots are supplied by deep nerves." This statement seems untenable anatomically and physiologically; Head puts down hair sensibility as "protopathic" and therefore cutaneous, and Boring, Trotter and Davies consider it distinctly cutaneous.

Burrow and Carter⁹ made unusually careful observations in 1,000 cases of peripheral nerve injury. They state that a common mistake is the use of too coarse stimuli which appeal to deep sensation when intended to stimulate the more superficial sense organs. They used an ermine hair brush for "light touch," and a spring algometer for "pin prick." With a piece of cork on this algometer thresholds of deep pressure sense were recorded. Thus their stimuli were nicely standardized. "Sharpness" was considered a form of epicritic sensibility. Thermal sense was examined only in spinal cord cases.

Price, Feiss and Terhune¹⁰ state that a brush and pin were used to test touch and pain respectively, and that "almost always the areas of impairment to both touch and pain are for all practical purposes the same."

7. Trotter and Davies: *J. Psychiat. u. Neurol.* **20**:123, 1913.

8. Souttar: *Brit. J. Surg.* **6**:279 (October) 1918.

9. Burrow and Carter: *Brit. M. J.* **2**:535 (Nov. 16) 1918.

10. Price, Feiss, and Terhune: *Arch. Neurol. & Psychiat.* **1**:547 (May) 1919.

Stookey¹¹ goes into the question of skin sensibility in peripheral nerve injuries very thoroughly, and is the first clinician to state boldly that to continue in the use of the terms "epicritic" and "protopathic" appears misleading. He wisely advocates the use of the specific terms "cotton wool area," "pin prick area," etc. Temperature tests were carefully made with known degrees of heat and cold. It is to be regretted that the "cotton wool" and "pin prick" were not equally well standardized, for many charts are published showing interesting dissociations of sensation which really cannot be interpreted because they lack these quantitative data.

This short review of the literature at hand brings out one point clearly—that the investigator should have in mind the object of his work, whether it is clinical diagnosis or physiologic investigation. All sensibility study is in a sense physiologic, but the physiologist must pay special attention to the distribution of specific end-organs, whereas these may be ignored by the clinician since the necessary cooperation on the part of the patient is not to be expected and simpler methods give all the facts necessary for diagnosis. The methods are so different that any compromise is useless. In fact, all who have been interested in the end-organs of cutaneous sensation agree that it is impossible to obtain accurate accounts of sensory phenomena from clinical subjects. So convinced were they of this fact that they all finally cut their own nerves for observation of these phenomena. Clinical men, on the other hand, although they usually come down to simple practical methods, do not have the courage of their conviction that these simple methods are sufficient, and having read some of the physiologic papers, cannot help dabbling in the complicated technic. Thus they spend time accumulating data that are of doubtful clinical value, and that would not be acceptable as physiologic evidence.

EXPERIENCES IN U. S. ARMY GENERAL HOSPITAL, NO. 11

In this clinic approximately 540 cases of nerve injury have been examined in the six months ending April, 1919, and the patients operated on, sixty-six in number, have been repeatedly examined by the ward surgeons and the writer. At first areas of dissociation were searched for and found. When the area of protopathic loss was smaller than that of epicritic, or when there was epicritic loss alone, the findings suggested that sensation was returning, or that the lesion was incomplete. But as the method of examination improved, faith in these distinctions waned; and with the advent of standardized

11. Stookey: Surg., Gynec. & Obst. 23:639 (Dec.) 1916.

algesiometers¹² and a standard method of examining for light touch¹³ no more areas of dissociation were discovered. For example, in a case of a recovering external popliteal lesion there was an oval area of disturbed sensation about 14 x 9 cm. When this area was examined with a needle prick of 30 gm., pain was everywhere felt, but when the pressure on the spring was reduced to 15 gm. the area of analgesia was found practically to correspond to that of tactile anesthesia. Thus a heavy prick elicits an area of dissociation and a light prick does not. If the usual method of testing with a pin held in the fingers had been used, the results would certainly have been variable and unsatisfactory.

Figure 1 shows a similar condition in a stationary lesion of the musculospiral nerve. But instead of finding no analgesia to the 30 gm. prick there is a smaller central area where even this strong stimulus is not felt. The lighter 15 gm. prick gives an area of analgesia practically corresponding to the anesthesia for light touch.

Figure 2, a musculospiral case with returning function, shows another common finding. The area for loss of pain to the 15 gm. needle prick is larger than the area where the brush is not felt, and the area for the 30 gm. prick is smaller. Thus we have three concentric areas, indicating that in these sensory examinations we are dealing with various grades of hypoaesthesia at the periphery, with anesthesia only at the center. Such findings would be impossible if there were two systems of cutaneous sensation which were either totally present or totally absent.

Figure 3 shows a similar condition in a case of complete division of the ulnar nerve, later checked up by operation. According to the older theories, the dissociation between the 30-gram-prick-line and the brush-line might have represented an area of returning sensation. Such examples could be repeated in large numbers. In fact, by vary-

12. The algesiometers used in this hospital were designed by Capt. S. D. Ingham. They consist of a sharp needle mounted on a spiral spring which plays back and forth in a brass tube the size of a small pencil. The compression of the spring can be regulated to make needle pricks of from 15 to 30 gm. pressure.

13. For "light touch" examination, a fine brush was employed, such as artists use for water colors. It was found necessary to pluck out about two thirds of the hairs, until the remaining pencil of hairs was so pliable that the skin could not be depressed by its application. Strokes with such a brush, at an angle to the surface of the skin, cannot elicit subcutaneous pressure sensations. Tampons of cotton wool, on the other hand, may become matted and exert enough pressure to stimulate subcutaneous end-organs. Such tampons are always too broad and clumsy to delimit accurately a small anesthetic area.

ing the quantitative values of the stimuli, dissociation of sensations can be predicted and produced almost at will. According to Trotter and Davies,⁴ if the exact locations of the pain spots are known, a prick of 1.5 gm. will elicit pain on normal skin, and very fine hairs will elicit touch sensation,¹⁴ but for practical purposes a soft brush that will not depress the skin may be used for cutaneous touch, or a needle of 15 gm. pressure for pain. These have been found by

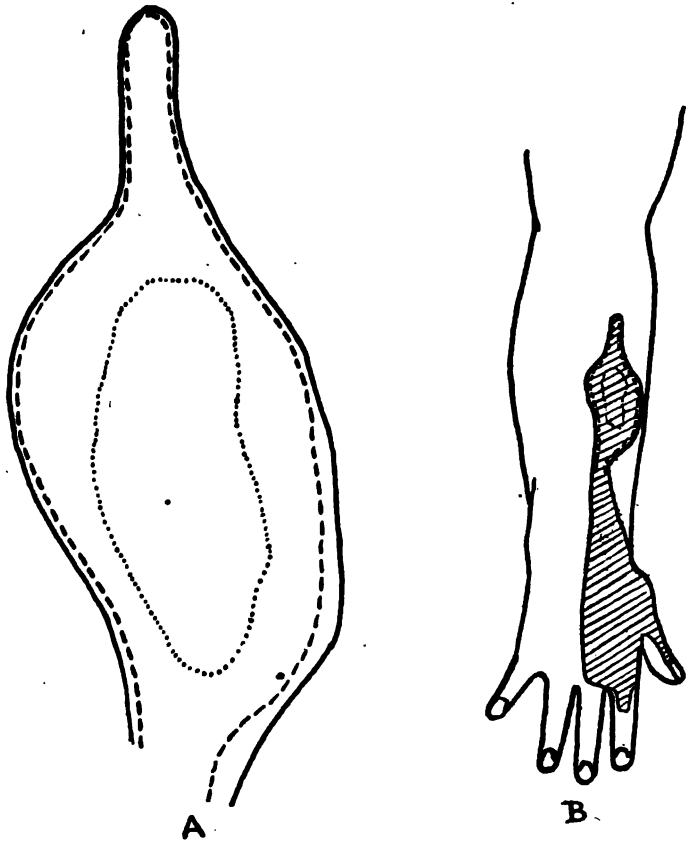


Fig. 1.—“A” represents a tracing ($\frac{3}{4}$ actual size) of the area of anesthesia of the patient mentioned in the text. The location of this area on the right forearm is indicated in “B.” The solid line is the boundary for the area of anesthesia for light touch with a brush; the broken line surrounds the area of analgesia for a needle prick of 15 gm., the dotted line for a needle prick of 30 gm.

experience to be practically equivalent stimuli; that is, they give almost co-extensive areas of anesthesia in any one case. As noted above, the same principle applies to thermal sense. It may be stated

14. Head and Rivers (Footnote 1); Trotter and Davies (Footnote 4).

therefore that "dissociations" of sensation in peripheral nerve lesions, arise from comparing stimuli not only qualitatively different but quantitatively unequivalent. In short, they are factitious because of lack of proper standardization of the methods of examination.

If these are the facts—and they seem to be proved by both experimental and clinical work—how should they affect the technic of examination? In the first place, it is obvious that some form of algometer should be used instead of a pin held in the hand. Also a standardized soft brush should be substituted for the tampon of cotton wool, and

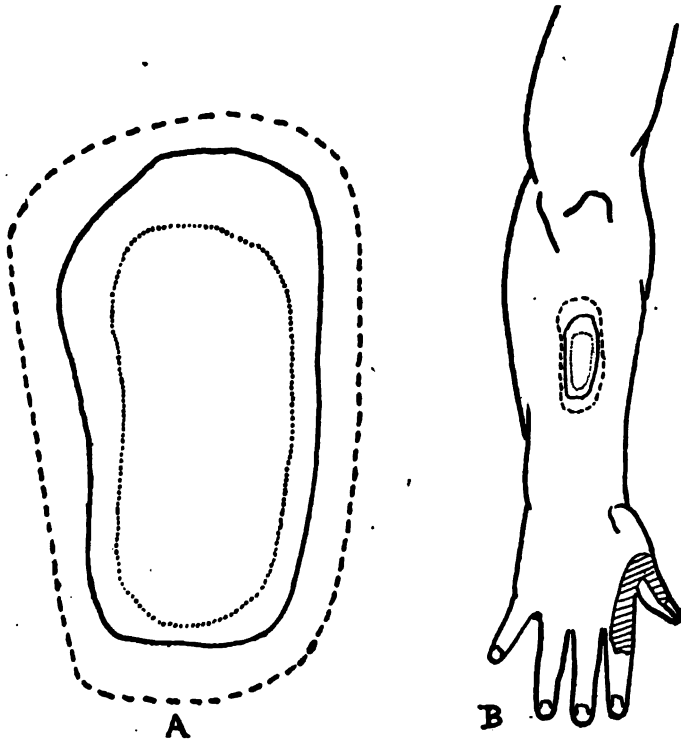


Fig. 2.—"A" and "B" represent a $\frac{1}{4}$ actual size tracing of the areas of anesthesia, and the location thereof, respectively. The symbols used are the same as in Figure 1.

known temperatures should supplant "hot" and "cold." In the second place, the fact that equivalent stimuli to different modalities of sensation give co-extensive areas of anesthesia makes it unnecessary to do more than one examination. But this one must be done carefully, and repeated with equal stimuli when later comparative examinations are made. Most clinicians use "pin prick"; Tinel,⁶ Dejerine and Mouzon,⁵ ask the patient to say whether the pin is felt as "touch" or "prick," thus outlining two zones. With cooperative patients this is

easy, but there are advantages in using the brush, for the patient merely has to answer "yes" quickly when he feels the stimulus.

It must be kept in mind that skin sensation is essentially punctate in distribution, so all methods that set out to find an area of anesthesia bounded by a sharp line are inaccurate. These boundary lines only indicate the average condition, showing nothing of the gradients between maximum and minimum sensitivity. "The method misrepresents a gradient by a line," as Boring puts it.⁵

In several cases pain has been brought out by a light needle prick well within the general area of analgesia. Careful testing showed

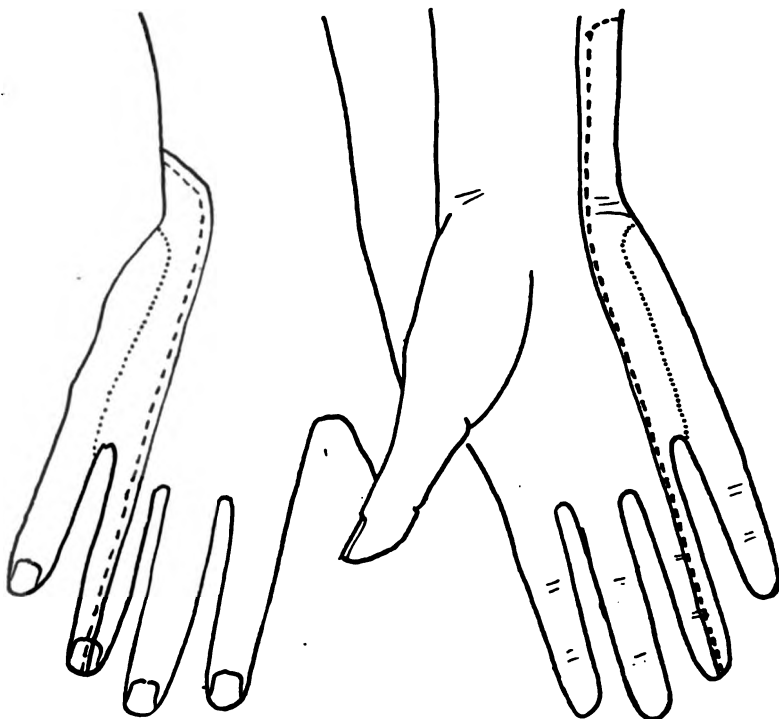


Fig. 3.—Areas of anesthesia as charted on a diagram of the hand. The solid line, broken line and dotted line represent the external boundaries of the areas of anesthesia for brush, 15-gram prick and 30-gram prick, respectively.

that these painful spots were usually along the course of a superficial vein. Trotter and Davies found that hyperalgesia persisted longer over such veins,¹⁶ than in other parts of the skin. This calls attention to the sympathetic system, and to the fact that some French authors have considered causalgia to be due to irritation of the sympathetic nerves.

15. Boring: *Quart. J. Exper. Physiol.* **10**:36, 1916.

16. Trotter and Davies (Footnote 4) Figure 7.

Possibly further investigation of the distribution of the hyperesthesia in patients with irritative lesions would be of interest in throwing light on this important subject.

In many peripheral nerve cases the paralyzed extremity is cold. Sensory examination on such extremities is unsatisfactory because the patient is not sure whether he really feels the tests or not, and gives variable answers which make it impossible to delimit sharply the area of anesthesia. In order to find out how important these varia-

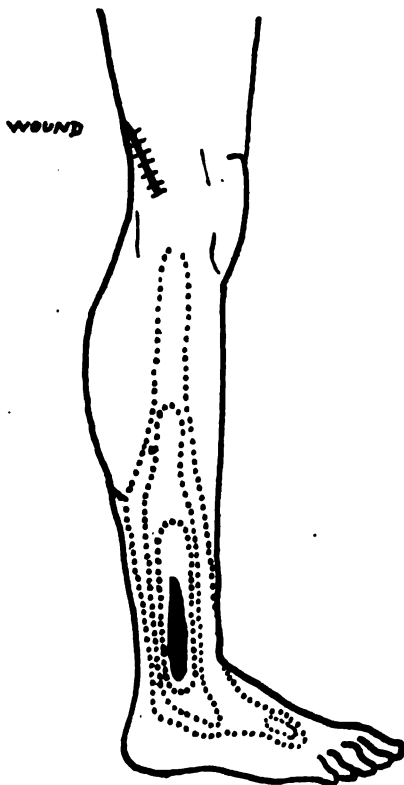


Fig. 4.—Diagram showing areas of anesthesia for varying grades of pressure. The solid black indicates anesthesia for 1,000 gm. pressure. From within outward, the next line shows the boundary of the area anesthetic to 800 gm.; the next, to 600 gm.; and the next, to 400 gm., as a contour map shows isometric altitudes. The case is one of complete interruption of the external popliteal nerve at the level of the head of the fibula.

tions might be several cases were examined, first with the extremity in its usual cold condition, and then after it had been warmed by immersion in hot water. Comparison of the results showed that the anesthetic area is larger when the hand is cold. In patients with

ulnar nerve lesions there was an error in the cold hand of from 0.5 cm. to 2 cm. and in the sciatics it even reached 5 cm. It therefore seems advisable to warm all cold extremities before examination. Burrow and Carter⁹ put the extremity to be examined into a paraffin bath and tested the skin while it was immersed in this liquid.

Subcutaneous pressure was tested with a spring instrument similar to the algometer, but with a blunt end about the size of a common pencil, and with a sliding scale on the side measuring pressures from 200 to 2,000 grams.¹⁷ Thus muscle and bone sensation can be tested in areas where the skin is anesthetic. The thresholds were recorded and charted in terms of grams pressure. Many such charts made interesting diagrams when the isometric points were joined by lines as in a contour map (Fig. 4). In general, the deep anesthesia seems to correspond to the muscles paralyzed, but much more work could be done in this line studying the anesthesia in relation to muscle innervation, and the overlapping of motor and of sensory innervations.

Vibratory and joint sensation has been found to be too grossly distributed to be of great value in localizing peripheral nerve lesions. A whole bone or a whole joint must be tested at once, and though the data obtained are interesting, they add little to the clinical diagnosis. Occasionally these tests help to distinguish an incomplete from a complete lesion, when the typical condition for a complete lesion is known.

Owing to the fact that most of the operations here have been done recently, there has been little opportunity to follow returning sensation in cases with known pathology. The experimental work reviewed above, of course, covered the general return of sensation, but it might be of interest to know how the areas of anesthesia usually receded for each typical sensory distribution. The fact that practical functional recovery often occurs while sensation is still largely impaired, makes it difficult and unfair to hold onto these patients. Stookey¹¹ and Dejerine and Mouzon⁶ show some charts of returning sensation; one median case was followed 281 days, but Head and Boring have found that the return is not complete after two years, even in small nerves.

CONCLUSIONS

1. A review of the experimental and clinical work on cutaneous sensibility indicates that the epicritic and protopathic hypothesis of Head and his collaborators should be abandoned.

17. This instrument was also designed by Captain Ingham and given to the writer to try out.

2. Dissociations of sensation due to peripheral nerve lesions arise from comparing stimuli not only qualitatively different but quantitatively unequivalent. In short, they are artefacts.

3. Clinical examinations should be simple, and since areas of dissociated sensation in peripheral nerve lesions are shown to be due to artefact, examination for one mode of sensation suffices for diagnosis.

4. For clinical sensory examinations quantitatively standardized stimuli should be used.

5. Subcutaneous pressure is best tested with an instrument which gives the threshold values in grams.

6. Hyperalgesia may follow the course of superficial veins.

SUPPLEMENTARY MUSCLE MOVEMENTS IN PERIPHERAL NERVE LESIONS

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INTRODUCTION

The frequency with which more than one muscle may produce a similar movement of the segments about a joint emphasizes the necessity for the use of great care in the analysis of all muscle movements. This care is the more necessary in the study of peripheral nerve lesions because the muscles under consideration may receive their nerve supply from different sources.

The preservation of certain movements the loss of which is supposed to follow particular nerve lesions has been observed for many years. Sherren¹ called attention to the fact that Swan, in 1834, was astonished at how much a rabbit could move its leg after experimental section of its sciatic nerve. Later Letievant² studied this phenomenon and termed it supplementary motility. Since that time numerous investigators have observed its presence in peripheral nerve lesions. To Duchenne³ and Beevor⁴ we owe much of the present knowledge of these movements. Sherren, Head and Sherren,⁵ Claude,⁶ and Athanassio-Benisty⁷ are among the recent observers who noted its presence.

These movements may be caused by a number of factors. Among these may be included the anastomotic supply of muscles from adjacent nerves, movements produced by muscles other than primary movers in this action, movements occurring as the result of mechanical factors producing a change of direction of leverage by shortening and lengthening of tendons and muscles passing over several joints, and slight movements resulting from the recoil of elastic tissue following a movement in a direction opposite to the one desired.

1. Sherren: *The Lancet* 1:727 (March 17) 1906.
2. Letievant: *Traites des sections nerveuses*, quoted by Sherren.
3. Duchenne: *Physiologie des mouvements*, Paris, 1867.
4. Beevor: *Croonian Lecture on Muscular Movements*, Adlar and Son, London, 1904.
5. Head and Sherren: *Brain* 28: Pt. 2, 116, 1905.
6. Claude: *Revue Neurologie*, April-May, 1916.
7. Athanassio-Benisty: *Clinical Forms of Nerve Lesions and the Treatment and Repair of Nerve Lesions*, Military Medical Manual, London, 1918.

SUPPLEMENTARY MOVEMENTS IN MUSCULOSPIRAL LESIONS

It is ordinarily understood that when the musculospiral nerve is divided there is lost, extension of the first phalanges of the fingers, extension of the wrist and of the thumb, adduction and abduction of the hand, and if the lesion is high, supination of the forearms when extended and, rarely, extension of the forearm.

The prime movers for extension of the distal phalanx of the thumb are the extensor longus pollicis, the abductor and adductor pollicis and the flexor brevis pollicis. Therefore, although never as complete or as strong as when the extensor longus pollicis is spared, the other muscles may produce extension of the distal phalanx of the thumb (Benisty*) (Fig. 1).

A simulation of this movement may be produced by flexion of the distal phalanx of the thumb followed by relaxation. Such a mechanism



Fig. 1.—Extension of the distal phalanx of the thumb in musculospiral palsy.

is frequently observed in slight flexion of the fingers, in ulnar and median lesions, and flexion and extension of the toes in internal and external popliteal lesions, respectively.

If by abduction of the thumb is understood that movement which carries the thumb away from the first finger in a plane at right angles to the palm, then my observations agree with Beevor in that the extensor ossis metacarpi pollicis is a prime mover, along with the abductor pollicis, opponens pollicis and outer head of the flexor brevis pollicis. Although in musculospiral palsy the patient is unable to carry the thumb away from the first finger in a plane parallel to the palm, abduction is possible in a plane at right angles to the palm.

In a musculospiral palsy it is possible to tense the proximal phalanges of the fingers by extending the terminal phalanges; at the same

time flexion of the proximal ones occurs, as the result of the unopposed action of the lumbricales. Slight passive extension of the proximal phalanges may be produced by flexion of the hand at the wrist. Simulation of extension of the first phalanx of the index finger is frequently accomplished by strong adduction and opposition of the thumb against the first phalanx of the index finger, which is thereby passively lifted dorsally.

Normally, extension of the wrist is accomplished by the extensors carpi radialis and ulnaris, extensor longus pollicis, and sometimes by the extensor communis digitorum.

In a lesion of the musculospiral nerve below the elbow, paralysis of the extensors of the fingers may occur without involvement of the extensors of the wrist. Under these conditions the patient cannot extend the wrist if at the same time he attempts to extend the fingers,

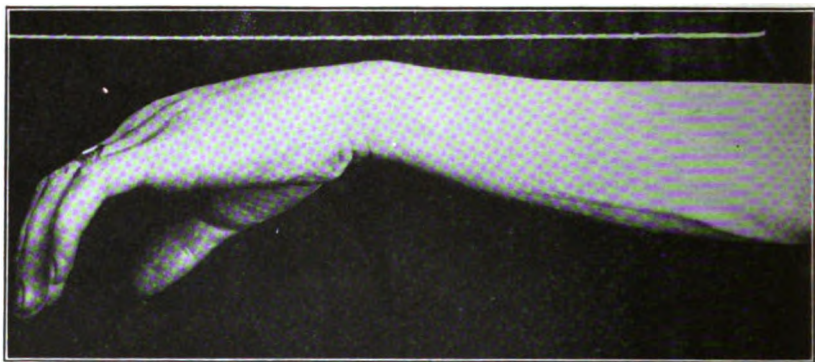


Fig. 2.—Wrist drop in musculospiral palsy.

but if he flexes the fingers extension of the wrist may then be accomplished (Duchenne³). To explain this we must recall the laws governing the action of muscles going over several joints. Beevor⁴ stated that "when a muscle by passing over two or more joints has two or more different actions, then if only one of these actions be required other muscles are brought into the movement whose actions are antagonistic to those of the muscles not required." These synergic muscles place the prime movers (in this instance being the extensors of the wrist) in the greatest elongation so as to augment their dynamic power and fix the joints so that the movements may be performed from a secure basis. Still another factor must be considered. Beevor has found that "if the movement of extending the wrist be performed with the fingers actively and fully extended, the extensors of the finger have to do all the work themselves and against the contraction of the flexors carpi

until the amount of work amounts to four or five pounds before the extensors carpi will join in and help them." In the cited instance of paralysis of the extensors of the fingers with preservation of the extensors of the wrist, the extensors of the fingers cannot possibly reach the amount of pull which is necessary before the extensors of the wrist can be made to contract.

In a lesion of the musculospiral nerve with paralysis of the extensors of the wrist dorsiflexion of the hand may be produced by the action of muscles not innervated by the musculospiral nerve. Dorsiflexion of the hand may occur in the course of energetic con-

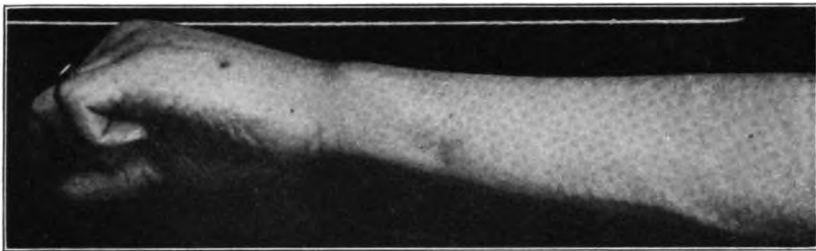


Fig. 3.—Passive extension of the wrist by flexion of the fingers.

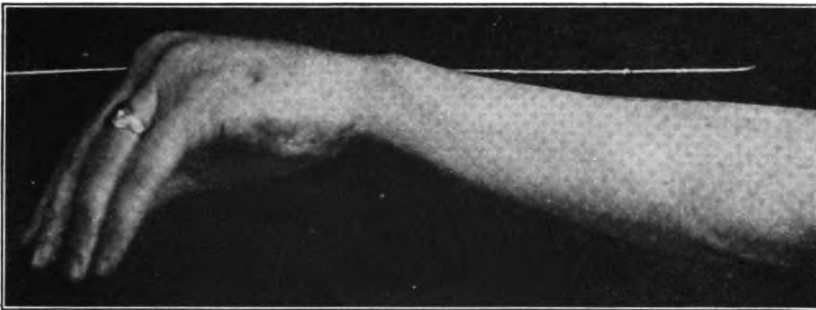


Fig. 4.—Passive extension of the wrist by strong contraction of the pronator radii teres.

traction of the flexors of the fingers. This occurs under certain conditions and has been noted frequently (Benisty⁷). When the wrist-drop does not exceed an angle of 120 degrees complete flexion of the fingers produces extension at the wrist. In this condition the extensors of the wrist are shortened by contracture and fibrosis so that the angle between the hand and forearm is such that passive dorsi flexion, or dorsal dislocation of the hand occurs when complete flexion of the fingers is accomplished. Without this provision the fingers could not be completely closed because of the shortened extensor tendons. The

mechanism may be illustrated by using the wrist as a hinge, the hand as the weight, the flexors as the power transmitted through a pulley at the metacarpophalangeal joint to a fixed point at the origin of the extensors of the wrist (Figs. 2 and 3).

In some cases strong contraction of the pronator radii teres will produce extension of the hand on the forearm. During this movement the head of the radius is strongly depressed toward the palm, the styloid process of the ulna is pulled dorsally and the hand is deviated to the ulnar side. It can be demonstrated readily that the hand can be flexed to a greater degree when the forearm is supinated

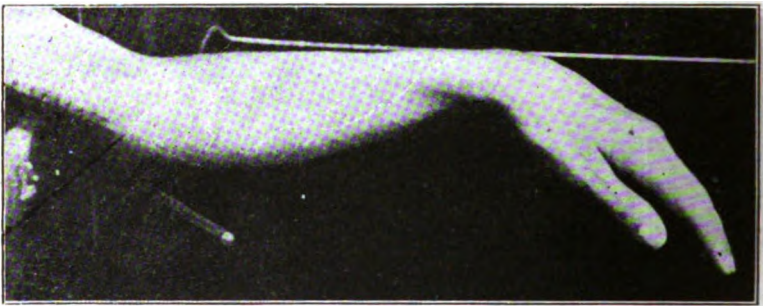


Fig. 5.—Wrist drop.



Fig. 6.—Passive extension of the wrist by adduction and opposition of the thumb against resistance of the index finger.

than pronated, and if flexed to its fullest degree when the forearm is supinated, the hand will be seen to extend when strong pronation is instituted. The extension at the wrist is probably due to two factors; first, lengthening of the extensor tendons and muscles, and second, to a leverage exerted on the scaphoid by the head of the radius (Figs. 2 and 4).

At times in addition to the contraction of the pronator, there is seen strong adduction and opposition of the thumb against the proximal phalanx of the index finger. At the same time resistance to this action

is made by the contraction of the lumbricale muscle, and the hand is extended on the forearm to a noticeable degree. During this action the middle, ring and little fingers show flexion at the proximal phalanx and extension of the two distal phalanges (Figs. 5 and 6). I have not found the last two mechanisms noted in literature.

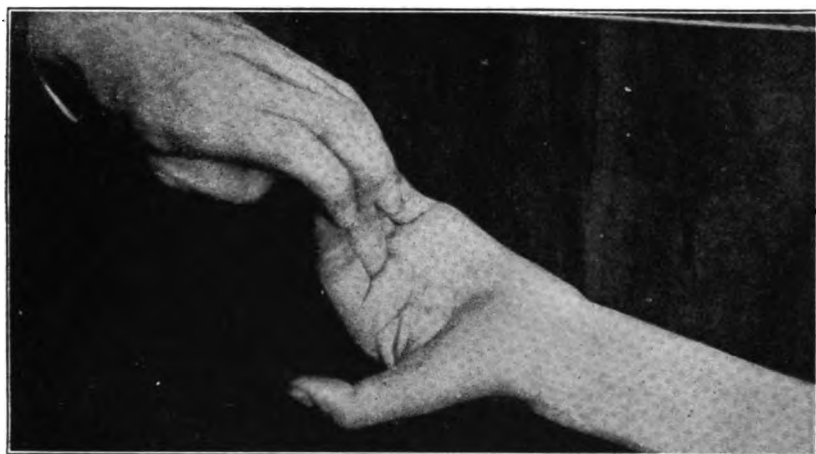


Fig. 7.—Tensing of the flexors of the fingers when strong flexion of the proximal phalanges is performed.



Fig. 8.—Opposition of the thumb by the adductor pollicis and the flexor brevis pollicis.

Supination with the forearm extended is performed by the supinator brevis. The action of this muscle is superseded by the external rotators of the shoulder, chiefly the infraspinatus, which carry out a movement weakly suggestive of supination. With the forearm flexed

the biceps is the most powerful supinator and in musculospiral paralysis supination is unaffected in this position.

Although Duchenne⁵ contended that abduction and adduction of the wrist were performed by the extensors of the wrist alone, and Benisty⁷ stated that the action of the flexor carpi radialis as an abductor and of the flexor carpi ulnaris as an adductor is negligible, I believe, as does Beevor and also McKenzie,⁸ that for pure lateral movements the extensors and flexors are both necessary. Adduction and abduction of the wrist are superseded by pronation and supination of the forearm in the position of wrist-drop of a musculospiral palsy. If, however, the hand be passively extended to the same plane as that of



Fig. 9.—Extension of the distal phalanges of the index and middle fingers in ulnar palsy.

the forearm, adduction accompanied by flexion of the wrist ensues as the result of contraction of the flexor carpi ulnaris, but I have not observed abduction to take place under the same conditions.

MEDIAN NERVE LESIONS

In a division of the median nerve it is supposed that the patient is unable to pronate the forearm, to tense the palm, to contract the flexor carpi radialis, to flex the second phalanges of any finger, to flex the distal or third phalanges of the index and middle fingers, to flex

8. McKenzie: *The Action of Muscles*, New York, 1918.

the proximal phalanges of the index and middle fingers, to flex the second phalanx of the thumb, to oppose or abduct the thumb, to fully flex the proximal phalanx of the thumb.

Supplementary movements to pronation have been described by Benisty,⁷ such as holding the elbow outward in flexion of the forearm, and rotating the arm inward in extension of the forearm.

Contraction of the palmar muscles and flexor carpi radialis cannot be supplemented.

Normally, flexion of the proximal phalanges is accomplished by the action of the lumbricales; flexion of the second phalanges by the flexor sublimis digitorum, and of the terminal phalanges by the flexor

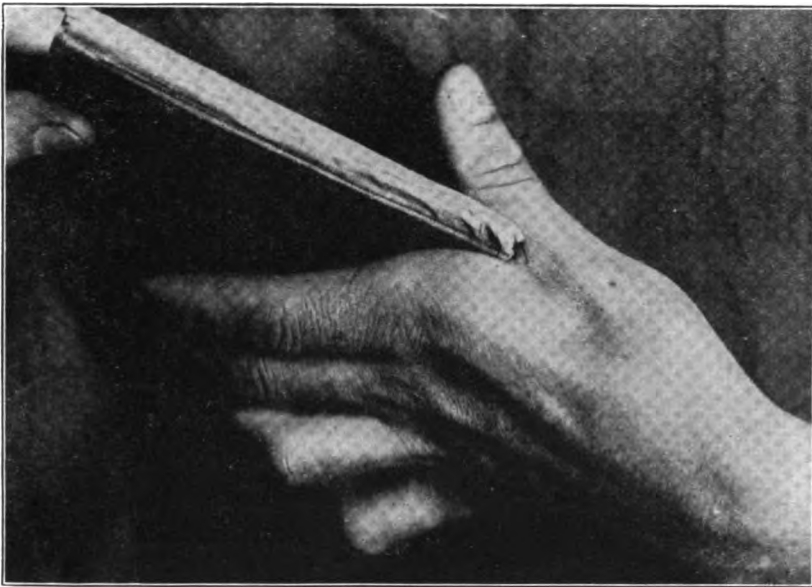


Fig. 10.—Adduction of the thumb by the extensor longus pollicis.

profundus digitorum. The flexor profundus digitorum for the two inner fingers, and the lumbricales for these fingers are supplied by the nerve.

Contrary to expectations, section of the median nerve frequently is followed by but little disturbance in the flexion of the proximal phalanges of the fingers. This seemingly paradoxical condition is due to a number of factors. Flexion of the proximal phalanges of the inner two fingers is preserved because the lumbricales of these two fingers are supplied by the ulnar nerve. The fact that the flexor profundus digitorum for the middle finger may in some instances receive its nerve supply from the ulnar, I think explains the frequent presence

of flexion of the first phalanx of that finger, inasmuch as the lumbricales have their origin in the tendon of the flexor profundus digitorum. If they are paralyzed, and especially if some contracture and shortening has taken place, contraction of the flexor profundus digitorum will produce a pull on the inert lumbricales and result in flexion of the proximal phalanx. That there is a pull exerted on the lumbricales seems to be shown by the fact that flexion of the first phalanx is stronger when combined with flexion of the terminal phalanges than when performed alone (Fig. 7). The lumbricale muscle for the middle finger likewise may receive its nerve supply from the ulnar. I am inclined to agree with McKenzie⁸ that the lumbricales flex the proximal phalanges and the interossei extend the distal phalanges of the fingers, under normal conditions. But as was seen to be the case with the flexors of the finger producing an extension of the hand, so under

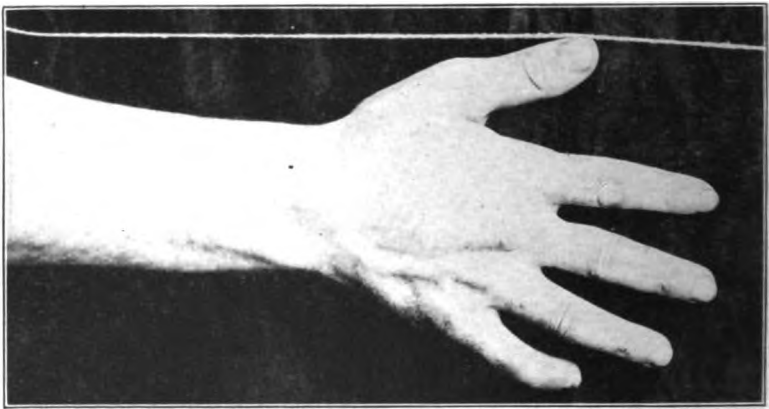


Fig. 11.—Abduction of the fingers by the extensor communis digitorum.

certain conditions the interossei may produce movements ordinarily subserved by the lumbricales. The interossei when extended produce a pull on the tendons of the flexors profundus and sublimis digitorum and when the lumbricales are paralyzed, especially if these are shortened, passive flexion of the proximal phalanges will occur. This mechanism permits full extension of the terminal phalanges, and in median nerve lesions occurs in the index and middle fingers. It is to be noted that despite the paralysis of the flexors in a median nerve lesion, the position of the fingers is one of flexion and not extension.

Flexion of the second phalanges of the inner two fingers occurs only a little weaker than normal as the result of the accompaniment of this movement to the normal flexion of the proximal and distal phalanges of these fingers. Flexion of the second phalanx of the middle

finger is frequently present in this general flexor movement. First, because it is influenced by flexion of the ring finger, and second, because the flexor sublimis digitorum for this finger in some instances must receive some of its nerve supply from the ulnar. Flexion of the terminal phalanx of the index finger is always absent. Flexion of the terminal phalanx of the middle finger may be present in those cases where the flexor profundus digitorum is supplied by the ulnar nerve.

Extension of the wrist produces slight passive flexion of the fingers which is better observed in combined lesions of the ulnar and median nerves.

Flexion of the terminal phalanx of the thumb may be simulated by the rebound following extension of this phalanx (Benisty⁷).

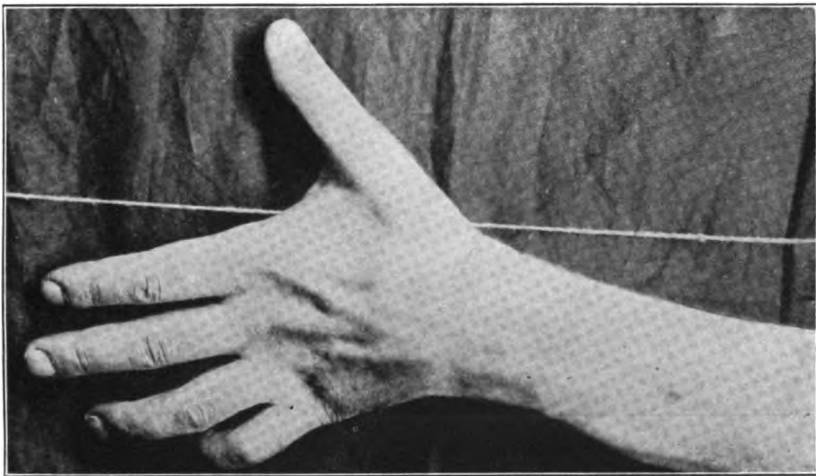


Fig. 12.—Abduction of the index finger by extension of the thumb and its metacarpal bone.

Opposition of the thumb may be simulated by the action of the adductor pollicis and the action of the adductor pollicis and the inner head of the flexor brevis pollicis, with the terminal phalanges of the finger being opposed, flexed (Fig. 8).

ULNAR NERVE LESIONS

Section of the ulnar nerve produces inability to flex the proximal or distal phalanges of the ring and little fingers, to abduct or adduct the fingers, to extend the second and distal phalanges of any of the fingers, to adduct the thumb, to contract the flexor carpi ulnaris, to abduct or oppose the little finger.

Flexion of the distal and proximal phalanges of the ring and little fingers is performed by the two inner tendons of the flexor profundus digitorum and the two inner lumbricales, respectively. The imperfect flexion of these phalanges is the result of influence exerted on all segments when the flexor sublimis digitorum contracts. This is more marked in the little than in the ring finger.

Slight flexion of the proximal phalanx of the ring finger may be obtained from the contraction of the flexor profundus digitorum pulling on the lumbricales muscles which has part of its origin from the tendon of the profundus.

Although the interossei which extend the second and third phalanges of all fingers are paralyzed, inability to extend these phalanges in the index and middle fingers is rare (Fig. 9). Benisty⁷ attributes this to the preservation of the lumbricales, which she states extend the second and third phalanges, as do the interossei. With this McKenzie disagrees.

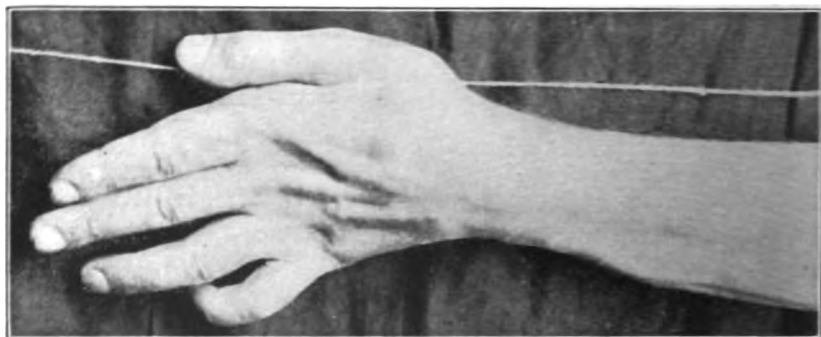


Fig. 13.—Adduction of index finger by extensor indicis with hand in ulnar deviation.

and he is inclined to believe that the dorsal interossei for the index and middle fingers receive some of their nerve supply from the median. Beside this, he believes that with hyperextension of the proximal phalanx, there is an alteration in the line of pull of the interossei which become angular instead of straight, and that an extended proximal phalanx forms a rigid dorsal support for the sublimus tendon, thus increasing its flexion pull. Therefore, paralysis of the lumbricales alone would produce at one time overaction of flexion of the second phalanx and a poor mechanical principle for extension of the distal phalanges.

I have observed preservation of part of the first dorsal interosseus several days following a resection and suture of the ulnar nerve. This was demonstrated by a distinct bellying of the muscle accompanying the movement produced by its contraction. This leads me to believe

that there is a dual nerve supply for the first and second dorsal interossei. Other factors, however, enter into the production of extension of the second and third phalanges of the index and middle fingers. Duchenne,³ Benisty⁷ and McKenzie⁸ contend that the extensor communis digitorum does not produce extension of these phalanges. On the other hand, Beevor⁴ pointed out that although it was true that when the extensor digitorum was paralyzed the second and third phalanges could be extended, and when the interossei were paralyzed claw hand occurred and extension of the second and third phalanges was impossible, yet if in the latter case the first phalanges were passively flexed, the second and third phalanges could be extended. He says it is probable that in claw hand the inability of the extensor digitorum to extend the terminal phalanges is due to its energy being expended on the first phalanges which are not prevented from overextension by the

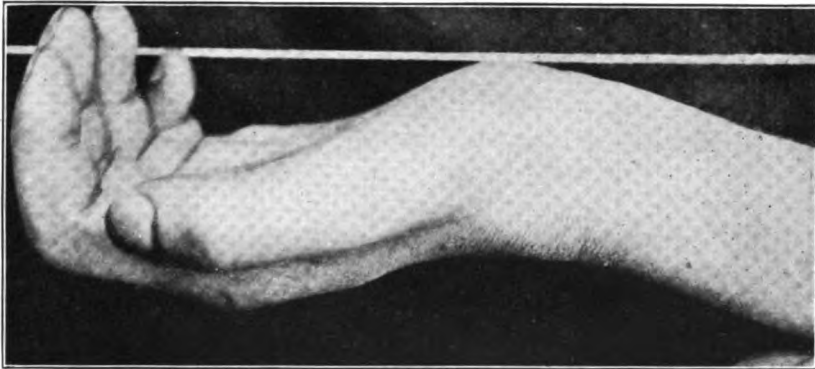


Fig. 14.—Position of wrist in combined ulnar and median lesion.

lumbricales which are paralyzed. I have been able to verify this in many cases of ulnar and combined ulnar and median nerve lesions.

The following factors enter into preservation of extension of the second and third phalanges: Innervation of the first and second dorsal interossei by the median, passive extension of the second and third phalanges by flexion of the proximal ones, thereby shortening the interossei. If the interossei are paralyzed and the lumbricales preserved, the pull on the interossei is straight and not angular; under these conditions, contraction of the extensor communis digitorum may exert a pull on the inert interossei and produce extension of the second and third phalanges. Some pull on the interossei may be exerted by the extensor communis digitorum even if these conditions are absent, as may be seen in combined ulnar and median nerve lesions.

The fact that the extensor communis digitorum exerts a pull on the inert interossei does not mean that it is at all concerned with the

normal extension of the second and third phalanges which may be the result of an entirely independent contraction of the interossei.

In adduction of the thumb, as pointed out by Duchenne,² the extensor longus pollicis is a prime mover, and in ulnar nerve lesions it may supplant the loss of the adductor pollicis (Fig. 10).

Abduction of the fingers away from the midline may result from forced extension of the first phalanges (Fig. 11). It is very marked in the index and little fingers. Slight adduction results from flexion of the first phalanges. Both of these movements have been known for a long time. The reason for the preservation of lateral movements in the middle and index fingers is given by Benisty⁷ as the preservation of their lumbricales, as well as the extensor action of the first phalanges.

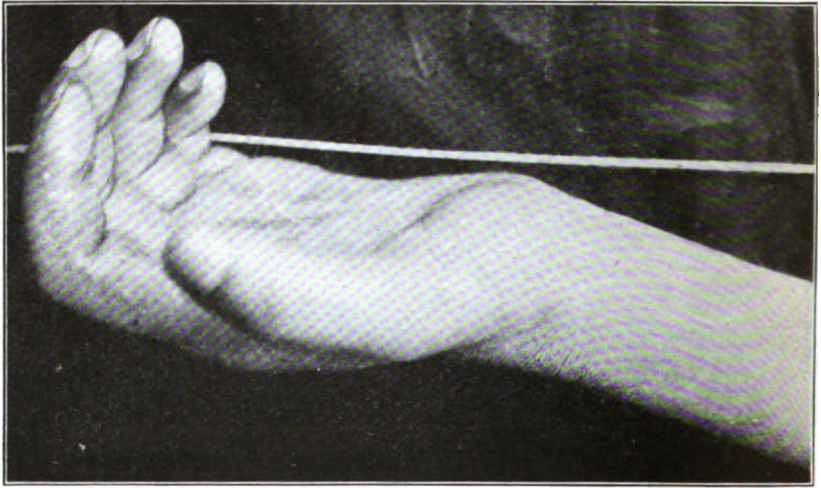


Fig. 15.—Flexion of the wrist by the extensor ossis metacarpi pollicis.

As McKenzie⁸ points out, the lumbricales are not concerned with lateral movements of the fingers. The preservation of lateral movements of these two fingers is due in addition to the extensor movements of the first phalanges, to a dual nerve supply, as it has been noted on a number of occasions that the first dorsal interosseus is partially preserved in complete ulnar section. Beside the abduction observed in forced extension of the first phalanx, abduction movement of the index finger can be produced by strong contraction of the extensor ossei metacarpi pollicis and extensor brevis pollicis; the first dorsal interosseus having part of its origin on the metacarpal bone of the thumb, is pulled onward by extension of this bone and produces abduction of the index finger (Fig. 12). When the hand is adducted to the ulnar side, the tendon of

the extensor indicis is so deflected that its contraction produces slight adduction of the index finger (Fig. 13).

In combined lesions of the ulnar nerves, Duchenne^a has pointed out that the extensor ossei metacarpi pollicis is a flexor of the wrist (Figs. 14 and 15).

SCIATIC NERVE LESIONS

In the lower extremity the principle supplementary movements have been observed in the toes as the result of rebound action following a movement in a direction opposite to the one intended.

In one case of a dissociated sciatic nerve lesion in which all the muscles supplied by this nerve with the exception of the flexors of the toes were paralyzed, strong flexion of the toes resulted in inversion and slight extension of the foot. The tibialis posticus was definitely paralyzed. This action was due to a mechanism similar to that observed in which strong flexion of the fingers produced passive extension of the hand musculospiral lesions.

MISLEADING MOTOR SYMPTOMS IN THE DIAGNOSIS OF NERVE WOUNDS *

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PHILADELPHIA

To the study of the neuromuscular system in peripheral nerve injuries, relatively insufficient thought is given, possibly because of the apparent crudeness of the problems involved. Yet in army hospital work we have found, in making decisions as to the operative and nonoperative treatment of nerve-wound patients, that motor symptoms have become our chief reliance. This is due to the fact that motor fibers are more vulnerable than sensory fibers, so that when mixed nerves are injured the earliest, and often the only, signs of trouble are presented by the motor fibers. Furthermore, from the patient's standpoint, motor defects are of much importance because of resulting disfigurement and inconvenience, and of the economic loss due to interference with work.

In dealing with peripheral nerve injuries the basal diagnostic fact on the motor side is that voluntary contractility in a muscle is proof that neural connection exists between it and its anterior horn centers. And, if disease of the muscle itself and of the corticospinal neurons need not be considered, the quality of the muscular contraction is a measure of the integrity of the motor nerve. If the opponens-pollicis, for instance, contracts poorly or not at all, we infer that the median nerve is proportionately damaged. In most cases these tests are easily applied and interpreted. But in gaging the condition of a nerve from the behavior of a muscle two causes of error are to be avoided: First, anomalous innervation occurs. If a given muscle is found to contract normally, we naturally expect to find its motor nerve uninjured. But several times in our experience the nerve which usually innervates the muscle in question has been found at operation completely severed, the motor axons to the muscle having reached it through another nerve. Some of the published cases in which function is said to have returned to a divided nerve immediately after suture are perhaps to be explained in this way: The nerve sutured was not the one that innervated the muscle under observation. Second, mistake is possible as to the fact of contraction in a muscle. The examiner supposes a certain muscle is contracting because of the movement in the limb which is ordinarily produced by that muscle. Closer inspection, how-

* Read before the Philadelphia Neurological Society, April 25, 1919.

ever, may reveal that the limb movement is being produced otherwise. In the diagnosis of nerve injuries in army hospitals these substitute movements were a prolific source of error. The following four classes of them are given as illustrations:

1. MOVEMENTS PRODUCED BY THE RESILIENCY OF NON-MUSCULAR TISSUES

When a muscle is normally innervated, a movement once willed proceeds from the original position of the joint continuously in the direction appropriate for that muscle. But when the patient tries to contract a paralyzed muscle, its antagonists being normal, he is apt to cause a preliminary contraction of the antagonists instead of the movement willed. Immediately following this momentary pull in the wrong direction, the joint is drawn back by the resilient soft tissues in the direction originally intended, just as if the paralyzed muscle had contracted. This phenomenon occurs oftenest in the interphalangeal

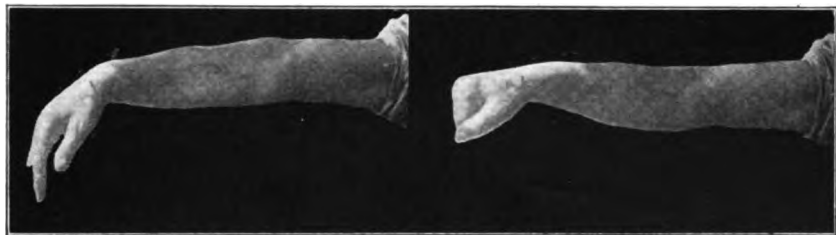


Fig. 1.—Musculospiral nerve severed. In the first picture the patient is trying to extend his wrist without flexing the fingers, but fails. In the second picture he has extended the wrist by flexing the fingers.

joints. To forestall the deception the joint should be held so as to prevent the contradictory movement of the antagonists, and the patient required to move the part from the start in the proper direction.

2. MOVEMENTS PRODUCED BY TENSION ON PARALYZED MUSCLES

When the tendon of a paralyzed muscle passes successively over two joints, the bending of either joint may place the dead muscle under tension and so cause the movement of the other joint which under normal conditions followed the contraction of the now paralyzed muscle. This was seen in our patients most often when there was paralysis of the long extensors of the digits (Figs. 1 and 2). When the fingers were strongly flexed, the extensor tendons were drawn over the knuckle joints and the whole extensor muscle put under tension. This pull on the unyielding muscle extended the wrist.

Dr. J. Ramsay Hunt¹ recently explained the extension of the wrist in a patient suffering from musculospiral paralysis as due to actual contraction of the extensor muscles. He assumed that this contraction was due, not to cortical impulses, but to impulses generated in the striate body and transmitted through hypothetical "paleokinetic" fibers that run in peripheral nerves and are able to struggle through obstructions that are impervious to ordinary motor fibers. In the "trick extension" described in the foregoing the extensor muscles of our patients were drawn palpably tighter, and the movement felt to the examining hand somewhat like a contraction; but the muscle-bellies did not show the characteristic shortening and bulging of an actual contraction. It seems possible that in Dr. Hunt's patient the extension may have been effected by the same mechanism as in ours.

The motor device under consideration was employed by patients in various other movements, such as extending the digits by actively flex-

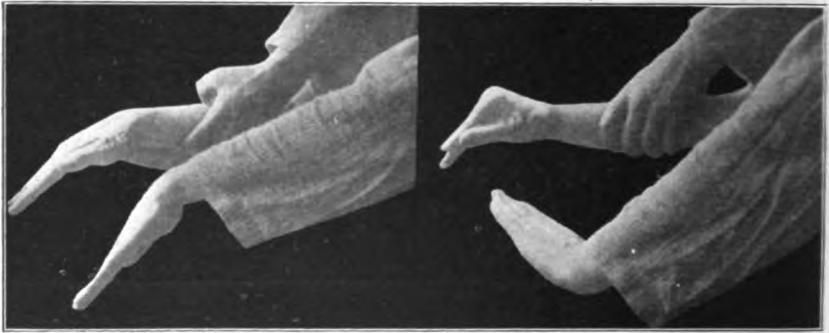


Fig. 2.—Right musculospiral nerve severed; long extensor muscles paralyzed. In the first picture the patient is about to attempt extension of the wrists. In the second picture he has succeeded with the left hand without disturbing the fingers; with the right, only by flexing the fingers and strongly contracting the *opponens pollicis*. (Passive tension on long extensors of thumb and fingers.)

ing the wrist when the extensors were paralyzed; and under the same conditions in extending the terminal phalanx of the thumb by active contraction of the *opponens* and *abductor brevis pollicis* (Fig. 3), and in flexing the digits by active extension of the wrist, when the long finger flexors were paralyzed (Fig. 4).

To eliminate this deceptive factor when testing a particular muscle, it is necessary only to prevent the patient from putting that muscle under tension. For instance, in testing extension of the ankle the toes must not be flexed, since that would tighten the extensor communis

1. *Brain*, September, 1918, p. 302 et seq.

and tend to extend the ankle. This precaution having been observed, any extension of the ankle that occurs is due to the *tibialis anticus* or its adjuvants.

3. SUBSTITUTES FOR MOVEMENTS OF THE INTEROSSEI

(a) Many writers² maintain that the *interossei* alone extend the interphalangeal joints. As this movement would thus offer a delicate test for the condition of the ulnar nerve (which supplies the *interossei*), the question is of practical importance. Following the teaching referred to, a patient's ability to extend the middle and distal phalanges has been repeatedly taken by surgeons as evidence that an injured ulnar nerve was regenerating, in spite of other evidence to the contrary. We became convinced, however, that the *interossei* were not the sole extensors of these joints by finding at operation in four patients



Fig. 3.—Right musculospiral nerve severed; long extensor muscles paralyzed. First picture: Effort to extend terminal phalanx of each thumb without disturbing proximal phalanx. Second picture: Succeeds with left thumb; fails with right thumb, but finally extends terminal phalanx of right thumb by contracting *flexor brevis pollicis* and *opponens pollicis*. (Passive tension on *extensor longus pollicis*.)

who retained this motion that the ulnar nerve was severed. In these men, furthermore, after the ulnar nerve had been resected and sutured the power to extend the phalanges was still present; neither before nor after operation was there any movement referable to the *interossei* (Fig. 5).

The only muscles capable of functioning as extensors of the interphalangeal joints except those in the ulnar nerve distribution are the

2. Benisty, A.: *Clinical Forms of Nerve Lesions*, p. 32. MacKenzie, William C.: *The Action of Muscles*, p. 116.

extension of the thumb, wrist, etc. It must therefore be true that the extreme proximal digital extensors, index and extensor digiti quinti, as well as the flexors, so that the presence of this paralysis does not necessarily indicate involvement or destruction of the entire nerve. In addition, it should be noted that both ulnar and median nerves are involved.

Fig. 4.—Paralysis of muscles controlled by left median and ulnar nerves.—The interosseus adduct and abduct the fingers, and these movements are a useful index of the condition

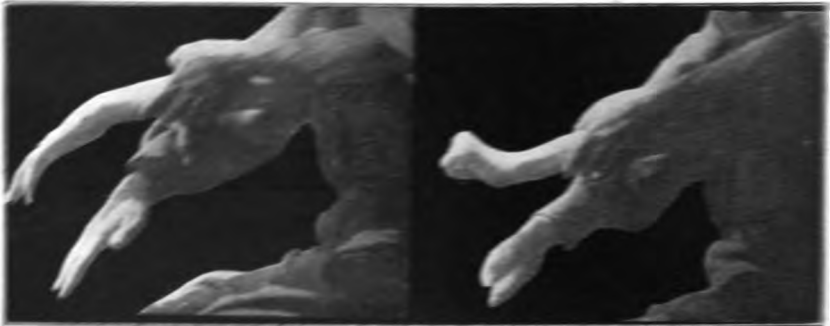


Fig. 4.—Paralysis of muscles controlled by left median and ulnar nerves. First picture: Attempt to flex digits without disturbing wrists. Second picture: Succeeds in the right hand, but flexes the left digits only by extending the wrist. (Passive tension on long flexor tendons.)



Fig. 5.—After surgical resection of left ulnar nerve; paralysis of interosseus. First picture: Metacarpophalangeal joints extended; inter-phalangeal joints flexed. Second picture: Contraction of long extensor muscles over-extends proximal phalanges, extends middle and distal phalanges.

of the ulnar nerve. Patients with ulnar nerve paralysis are, however, able to separate their fingers by extending them, because the direction of the pull of the long extensor tendons is such that it brings each phalanx into line with its metacarpal (Fig. 6). For this reason abduction and adduction should be tested with the metacarpophalangeal joints slightly flexed. A convenient test is to have the patient place his finger tips and wrist on a smooth surface. A coin is then placed

under the finger-tips one at a time, and the patient attempts to move it laterally. If he succeeds in doing this with each finger while the other fingers and the base of the hand are kept firmly in position, it is positive proof that the interossei are functioning.

4. ERRORS IN INTERPRETING MOVEMENTS OF PRONATION, SUPINATION AND FLEXION OF THE FOREARM

Pronation is ordinarily accomplished by the pronator teres and the pronator quadratus, innervated through the median nerve. Several of our patients with median nerve paralysis were, nevertheless, able to pronate by another device. The long wrist and finger extensors and the brachioradialis pursue a spiral course from the external condyle to or beyond the dorsum of the wrist. Contraction of this group therefore starts pronation and carries it half way. The patient then flexes his wrist and gravity carries the hand into complete pronation.

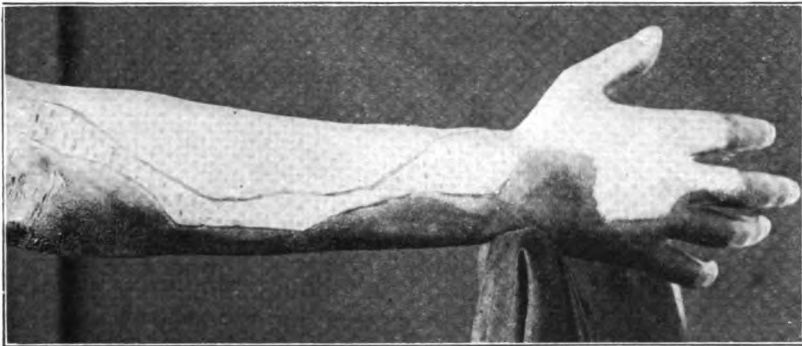


Fig. 6.—Ulnar nerve severed; interossei paralyzed. Patient is in the act of extending his fingers. The direction of the pull of the extensor tendons and the conformation of the surfaces of the metacarpophalangeal joints cause the fingers to separate.

Supination, if normally performed, shows that the musculospiral (supinator brevis muscle) and musculocutaneous (biceps muscle) are intact. The statement has been made that the biceps cannot aid in supination unless the elbow is partly flexed, and in one of our cases the question became of some importance. The patient had a serious musculospiral injury for which operation seemed advisable; but because he could supinate while the elbow was extended one surgeon counseled against operation, claiming that the supinators and the musculospiral nerve were acting. In this patient, also in several others with complete loss of musculospiral function, the findings at operation demonstrated that the biceps supinates the forearm even when completely extended.

Flexion of the elbow is dependent on the musculocutaneous (biceps and brachialis anticus) and musculospiral (brachioradialis). Yet one patient in our series with paralysis of both musculospiral and musculocutaneous nerves was able partly to flex his elbow, at the same time pronating it, by contracting his pronator teres. He had used the muscle so constantly in feeding and dressing himself that it was hypertrophied, and stood out conspicuously above the other forearm muscles.

THE EYE SYMPTOMS IN PSEUDOTUMOR CEREBRI, WITH REPORT OF AN ADDITIONAL OBSERVATION.

RETROCESSION OF BILATERAL CHOKED DISK TO NORMAL; UNILATERAL
DEAFNESS FOLLOWED ONE YEAR LATER BY THE LOSS OF THE
VESTIBULAR FUNCTION AND THE UNILATERAL APPEAR-
ANCE OF THE ROMBERG SIGN

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DEFINITION

By pseudotumor cerebri we understand a syndrome that presents all the cardinal symptoms of brain tumor, a syndrome which, in fact, does not admit of any other diagnosis, yet the consequent clinical course of the disease, whether it leads to improvement or to a cure, does not confirm the primary diagnosis. A certain number of necropsies following an operation or sudden death yielded entirely negative results, since no neoplasm in the brain could be found. In about ten cases certain alterations were found, but these were of so slight a nature that they could by no means explain the symptoms, and in three cases, even with the most exact microscopic examinations of the brain, no cause could be discovered for either the disease or its fatal termination.

HISTORY

Hughlings Jackson¹ published in 1876 a report of a case with all the symptoms of brain tumor and double choked disk, in which the necropsy gave an entirely negative result, since only a slight congestion of the brain was found. In 1891 Eichhorst² published an account of his observations which proved that meningitis serosa may produce the same symptoms as brain tumor. Byrom Bramwell³ published in 1899 "some cases of distended ventricles simulating a cerebral or cerebellar tumor," in which neither operation nor necropsy could detect a neoplasm. Wernicke observed the same symptoms in encephalomalacia, and the observation has since frequently been made by neurologists

1. Jackson, J. Hughlings: Royal London Ophthalmic Hospital Reports, 1876. 1876.

2. Eichhorst: Hydrocephalus internus idiopathicus bei Erwachsenen, Ztschr. f. klin. Med. 19: 1891, Supplement.

3. Bramwell, Byrom: On the Localization of the Intracranial Tumors, Brain 22:66, 1899.

and especially by brain surgeons (Krönlein, Küttner and others). This observation is all the more surprising for the reason that necropsies did not show an increase of the brain volume in proportion to the size of the cranial cavity. It is by this disproportion that the origin of the most outstanding symptoms of the brain tumor can be explained. Oppenheim, in 1901, called attention to the fact that great care should be taken in the diagnosis of brain tumor in children who present the general symptoms of this condition, but accompanied by signs referable to the motor cortical area. Nonne,⁴ who had especially observed and studied such cases, came, in 1904, to the following conclusions, based on eighteen observations: "There are clinical syndromes which, in the light of our present-day experience and limited knowledge, justify us in making the diagnosis of brain tumor, though the subsequent clinical course may show us that the diagnosis was wrong and retrospective reflection may not make clear to us the course of our diagnostic error. I refer to the cases which ended in a cure as well as to those followed by negative necropsies." Among Nonne's cases were found some in which the most exact microscopic, histologic and bacteriologic investigation failed to show anatomic alteration or infection. His observations were very soon followed by others, so that we have today about ninety cases in the literature. Mohamed Saleh⁵ gathered in 1912 all the cases published prior to that year. From his critical comparison most of the following statements are borrowed.

CLINICAL ASPECT

Mohamed Saleh discusses sixty-three cases reported in the literature and divides them into four clinical groups:

1. Symptoms of brain reaction without convulsions or signs of localization. (Ten cases with eight necropsies.)

All these patients presented headache, vomiting, vertigo, torpor cerebri, choked disk and ictus without convulsions. One case showed retardation of the pulse (30 beats a minute); two cases showed loss of weight. The deep and superficial reflexes were normal.

2. General symptoms with convulsions (fourteen cases with eight necropsies).

These cases showed epileptiform fits which were in some instances the first symptoms, and in others the last.

4. Nonne: Ueber Fälle vom Symptomenkomplex Tumor cerebri mit Ausgang in Heilung (Pseudotumor cerebri); über letal verlaufende Fälle von Pseudotumor cerebri mit Sektionsbefund, *Deutsch. Ztschr. f. Nervenhe.* **27**: 169, 1904.

5. Saleh, Mohamed: *Etudes critiques des états dits: pseudotumeurs cérébrales*, Thèse de Lyon, 1912.

3. General symptoms without epilepsy, but with signs of cortical localization (nineteen cases with seven necropsies).

These cases showed, besides intracranial hypertension, signs of unilateral cortical localization, such as monoplegia, hemiplegia, hemianopia, hemianesthesia, aphasia, alexia and ear ailments.

4. Cases with cerebellar symptoms (twenty-three cases, nine necropsies; three of the cases controlled during operation).

These cases showed asthenia, lateropulsion and cerebellar gait.

Pseudotumor shows no preference as to sex or age. Among the thirty cases collected by Mohamed Saleh in which necropsies failed to discover a neoplasm, there were fifteen men, thirteen women and two children. The youngest was 10 years old, and the oldest 79; but we may conclude that pseudotumor cerebri, like genuine brain tumor, is largely a disease of mature years, since only five out of thirty patients were under 20 years of age. Pseudotumor differs from genuine brain tumor in that the latter predominates in the male sex. Martin⁶ found in 513 cases of intracranial tumor 70 per cent. males and 30 per cent. females.

ANATOMIC FINDINGS

Necropsies were performed in thirty-two of Mohamed Saleh's cases. The alterations were in most cases minimal; in thirteen they were found almost inappreciable. Among the other nineteen there were found ten cases with hydrocephalus internus, four with encephalitis hemorrhagica, one with encephalomyelitis diffusa, one with meningitis chronica basalis, one with empyema ventriculare, and two with encephalomalacia. All of these cases had in common an abundant serous exudation in the brain with resultant increase of the intracranial pressure. Therefore, it may be assumed that the increased intracranial pressure, together with the toxoinfection, produced the general as well as the local symptoms.

DIFFERENTIAL DIAGNOSIS

The diagnosis of pseudotumor cerebri can only be made with absolute certainty by a necropsy. We know from the thesis of Mohamed Saleh that many diseases are accompanied by the symptoms of brain tumor, and that some of these diseases have as yet an obscure symptomatology. The diagnostic difficulties arise in cases that result in a cure. In this connection the following data may be presented:

1. There are records of brain tumor discovered accidentally by necropsy which during life had not shown noticeable symptoms

6. Martin: The Localizing Value of Optic Neuritis in Intracranial Tumor, *Lancet* 2:81, 1897.

(Eichhorst). Thus, Byrom Bramwell reported a case in which a cerebellar tumor became encapsulated and, as a result, ceased to produce symptoms. Williamson and Roberts observed a myxoma of the cerebellum that had not shown symptoms during forty-six years. There are further records of benign tumors (psammomata, lipomata, cholesteatomata, aneurysmata, tuberculomata and echinococcus, in which the brain, after a state of intolerance, became accustomed to these growths. But these cases are very rare, and they show almost always residual symptoms, such as constant headache or epileptiform fits, even if all other symptoms disappear.

2. Hydrocephalus internus adutorum may be produced by all sorts of cachectic conditions. Eichhorst states that it can be found in pulmonary tuberculosis and chronic nephritis, as well as in saturnism or chlorotic anemia (Patrick⁷). It is of special importance that internal cachectic hydrocephalus can simulate, for instance in ventricular carcinoma, an apparent metastasis in the brain. Hydrocephalus internus is furthermore frequently produced by infection of the ependyma which may have its origin either in the external layer of the brain, as in purulent, tuberculous or syphilitic meningitis, or through the blood. Pierre Merle⁸ in his thesis has shown experimentally that acute infection of the ependyma gives the clinical aspect of meningitis, and that a chronic infection produces all the symptoms of a brain tumor. We must further remember that myxedema may produce in adults transient internal hydrocephalus which recedes on thyroid treatment, and that similar symptoms may be produced by pregnancy, likewise receding on its interruption. Nolen⁹ has observed an excellent example of this:

CASE 1.—A patient who fell ill during the second half of her twelfth pregnancy had headache, vomiting, torpor cerebialis and hemiparesis of the right side. After the birth of the child all symptoms receded. The thirteenth pregnancy terminated in the fourth month without brain symptoms. In the third month of her fourteenth pregnancy she again fell ill, having the same brain symptoms as before, accompanied by bilateral choked disk and unilateral total reflex immobility of the pupil. A pulmonary infection made artificial abortion necessary. Several days later there appeared a left-sided ptosis and paralysis of the left internus; following this the symptoms slowly receded. Three years

7. Patrick: Brain Tumor Simulated by Anemia, *J. Nerv. & Ment. Dis.* 25:882, 1898.

8. Merle, Pierre: *Etudes sur les épendymites cérébrales*, Thèse de Paris, 1910.

9. Nolen: Ueber einen Fall von Pseudotumor cerebri, der im Verlauf einer Schwangerschaft auftritt u. in zwei folgenden Schwangerschaften recidivirt, *Berl. klin. Wchnschr.* Dec. 6 and 13, 1909.

later in the eight month of another pregnancy, the same symptoms appeared. Within two months after the birth of the fifteenth child all symptoms disappeared.

Nonne believes that in cases which result in recovery we can exclude internal hydrocephalus as the cause when the history does not show chronic alcoholism, physical or psychic trauma insolation, or tuberculous or syphilitic infection. A good example of the influence of a psychic trauma has been observed by Nonne:

CASE 2.—A young man, aged 30, who had always been healthy, suddenly heard that his wife had been crushed in a railway accident. He instantly fell ill, became weak and vomited. The following day he complained of headache, which became so violent that he went to the hospital. An examination revealed: pulse 36, pupils dilated without reaction and a bilateral choked disk. He died within a few days, and necropsy disclosed nothing beyond slight hyperemia of the ependyma and slight internal hydrocephalus.

Hoppe¹⁰ states that even then we cannot exclude hydrocephalus with certainty, because hydrocephalus may also be produced by other causes unknown to us. He reminds us that acquired hydrocephalus may occur from inflammation of the choroid plexus from pressure on the vena magna Galeni, or localized inflammation around the opening of the fourth ventricle.

So far as encephalomeningitis is concerned, we can never be absolutely sure of the diagnosis, because we cannot rule out with certainty either localized tuberculous or localized syphilitic meningitis. We know by the observation of Eichhorst that tuberculous meningitis may heal either spontaneously or after lumbar puncture. Martin¹¹ cites twenty cases of healed tuberculous meningitis from the literature. We know by the observation of Rosenfeld¹² that nonsuppurative encephalitis may heal spontaneously.

Furthermore, we must remember also that serous meningitis may cause hemiplegia, aphasia and cerebellar ataxia, and that local symptoms are not always produced by local anatomic alterations. Quincke,¹³ who worked especially on serous meningitis, records that in the acute form fever may be absent, but that somnolence and rigidity of the neck are always present. He further says that the chronic form may have for years the aspect of a slowly growing brain tumor, showing nothing

10. Hoppe: Brain Tumor Symptom-Complex with Termination in Recovery, *J. Nerv. & Ment. Dis.* 34:97, 1907.

11. Martin: The Occurrence of Remissions and Recovery in Tuberculous Meningitis; a Critical Review, *Brain* 32:209, 1909.

12. Rosenfeld: Encephalitis der optischen Bahnen, *Centralbl. f. Nervenhe.* 23: 1905.

13. Quincke: Ueber Meningitis serosa u. verwandte Zustände, *Deutsch. Ztschr. f. Nervenhe.* 9: 1897.

more than headache, vertigo and alterations of character in the form of neurasthenia. Serous meningitis develops in most cases between the ages of 5 and 30. The younger the patient, the more acute is the form, and it generally follows an infectious disease like typhoid or pneumonia. The presence of choked disk argues more in favor of serous meningitis. Cushing and Bordley¹⁴ emphasized that meningitis of infectious origin very seldom conduces to choked disk.

PROGNOSIS

About half of the cases end in recovery. In some cases death comes very suddenly and necropsy furnishes no explanation. Some patients suffer one or more relapses. Of special ophthalmologic interest here are the cases of Hoppe, Higier, Finkelnburg, and Dor and Jaboulay, which showed recurrence after years of complete health, and in some of the cases three times, with a newly appearing choked disk. It is also remarkable that some patients die surprisingly soon after an exploratory operation.

THERAPY

It is obvious after the foregoing description that in any case showing symptoms of brain tumor we should follow the advice of Horsley, to treat the patient at least during six weeks with strong doses of iodine, and, if there are anamnestic indications, to use also mercury, arsenic, thyroid extract, etc.

It is further advisable to do a lumbar puncture, but it must be done with precaution. Trocme¹⁵ cites from the literature of the subject thirty-five cases in which death followed lumbar puncture. The precautions may be summed up in the following points: The patient should lie down for half an hour before the operation. The puncture should always be done with patient in a lateral position, the head being kept a little lower than the feet. The withdrawal of fluid should be made slowly and in not too large a quantity. It should be interrupted if the patient complains of increased headache or feels weaker. The patient should continue in a horizontal position for some time after the lumbar puncture has been completed. On the other hand, we call attention to the fact that Quincke has already pointed out the curative effect of the lumbar puncture in meningitis serosa.

As a second step, later on, craniectomy can be done, without incision of the dura. Thus, Henri Claude and F. Lejars¹⁶ made the

14. Cushing, H., and Bordley, J.: Observations on Choked Disk, *J. A. M. A.* 52:353 (Jan. 30) 1909.

15. Trocme: De la thérapeutique palliative dans les tumeurs de l'encéphale, Thèse de Paris, 1909.

16. Claude, Henri, and Lejars, F.: Deux cas de méningite séreuse localisée de la région cérébelleuse et protuberantielle, *Société médicale des hôpitaux de Paris* 2:817, 1913.

diagnosis of pseudotumor in two cases in which all the symptoms disappeared after craniectomy without incision of the dura. They mention that sometimes cysts of the arachnoid, produced by adhesive arachnoiditis or by a hemorrhage are found instead of true neoplasms. Such cysts are more easily discovered during operation than through necropsy, when the walls rupture during the opening of the skull. Similar cysts over the convexity of the brain have been found by Wendel,¹⁷ Zesas,¹⁸ Bachelier¹⁹ and Mehmed.²⁰ They have been found in the cerebellopontine angle by Placzek and Krause,²¹ Unger²² and Finkelstein²³ and Alfred Murray.^{23a} Sicard holds that in any case we can make the diagnosis of pseudotumor, if the symptoms definitely recede after palliative craniectomy, because numerous statistics show that palliative operation cannot prolong a patient's life longer than eighteen months in true neoplasm of the brain.

EYE SYMPTOMS

In the presence of headache, vomiting and vertigo, the diagnosis of brain tumor is chiefly made on the appearance of choked disk. It may therefore be advisable to give a brief summary of the eye symptoms in cases of pseudotumor cerebri.

A. CASES CONTROLLED BY NECROPSY

If we accept as positive cases of pseudotumor cerebri only those in which necropsy failed to reveal neoplasm in the brain, we would have to consider thirty-two cases mentioned in the thesis of Mohamed Saleh. To these we may add two cases mentioned in the paper of Byrom Bramwell; the case of Hughlings-Jackson; two cases of Williamson and Roberts,²⁴ who found, out of one hundred cases of

17. Wendel: Ueber Meningitis serosa circumscripta cerebri, Arch. f. klin. Chir., 1912.

18. Zesas: Ueber Meningitis serosa externa circumscripta cerebri, Samml. klin. Vortr. auf d. Gebiete d. Chir., 1913, No. 188.

19. Bachelier: La meningite séreuse circonscrite de la corticalité cérébrale, Thèse de Lyon, 1913.

20. Mehmed: Sitzung der Herzte des Gulhane Krankenhauses in Konstantinopel, 1913, Deutsch. med. Wchnschr., 1914, p. 576.

21. Placzek and Krause: Zur Kenntnis der umschriebenen Arachnitis adhäsiva cerebri, Berl. klin. Wchnschr. 22: (July) 1907.

22. Unger: Ein operirter Fall v. Arachnitis circumscripta syphilitica der hinteren Schädelgrube, Berl. klin. Wchnschr. Feb. 1, 1909.

23. Finkelstein: Russk. Vrach., 1908.

23a. Murray, Alfred: A Case of Bilateral Choked Disk Produced by a Cyst of the Cerebello-Pontine Angle; Successfully Operated on: Restoration of Vision, Ophth. Rec. 20:245, 1911.

24. Williamson and Roberts: Remarks on the Diagnosis and Prognosis in 100 Cases of Double Optic Neuritis with Headache, Lancet 1:1350, 1900.

double optic neuritis with headaches, two instances of slight internal hydrocephalus; further, one case each of Diller;²⁵ Riggs;²⁶ Gowers, Kupferberg,²⁷ and Engelhardt;²⁸ Pouseppe²⁹ records three cases with negative necropsy, but the reference that was at my disposal was too brief.

Out of these forty-six cases, confirmed by necropsy, forty-three, whose history I was able to read, showed:

1. Choked disk in varying degree.

(a) Present in thirty cases: Of these, twenty-eight were bilateral; namely, nine cases of Nonne;³⁰ one case each of Hoppe, Long,³¹ Sicard,³² Reichhardt,³³ Schroeder,³⁴ Vincent, Marinesco,³⁵ Ramsay Hunt,³⁶ Rosenfeld, Weber and Schultz,³⁷ Hughlings-Jackson, Diller, Riggs, Gowers, Kupferberg, Engelhardt; two cases of Byrom Bramwell and of Williamson and Roberts, and one case of Finkelnburg and Eschbaum,³⁸ which showed postneuritic optic atrophy in both eyes without restriction of the visual field as residuum of the choked disk. In two cases the choked disk was unilateral (cases of Nonne and Devic-Saleh).

25. Diller: A Case of Serous Meningitis Simulating Brain Tumor, *J. Nerv. & Ment. Dis.* **25**:441, 1898.

26. Riggs: Symptoms Simulating Brain Tumor Due to the Obliteration of the Longitudinal, Lateral and Occipital Sinus, *J. Nerv. & Ment. Dis.* **34**:260, 1907.

27. Kupferberg: *Deutsch. Ztschr. f. Nervenhe.*, 1893.

28. Engelhardt: Neuritis Optica bei Chlorosis; *Krankheitsverlauf u. Tod unter den Symptomen eines Hirntumors*, Münch. med. Wchnschr., 1900, p. 1233.

29. Pouseppe: *Russian Rev. of Neurol.*, 1913; abstr. *Rev. neurol.*, 1914.

30. Nonne: Beiträge z. Frage v. Pseudotumor cerebri, *Deutsch. Ztschr. f. Nervenhe.* **27**:169, 1904; *Deutsch. Ztschr. f. Nervenhe.* **33**:317, 1909; *Neurol. Centralbl.*, p. 961, 1907. Differentialdiagnose des Tumor cerebri, *Neurol. Centralbl.*, July 15, 1912.

31. Long: Transactions of the Société de neurologie, *Rev. neurol.*, Jan. 12, 1911.

32. Sicard: Transactions of the Société de neurologie, *Rev. neurol.*, Jan. 12, 1911.

33. Reichhardt: Entstehung der Hirndrucksymptome, *Deutsch. Ztschr. f. Nervenhe.*, p. 339, 1905.

34. Schroeder: *Hospitaltid.*, June 16, 1909.

35. Marinesco: Deux cas de pseudo-tumeur cérébrale: méningite séreuse et hydrocéphalie acquise, *Nouv. iconog. de la Salpêtrière*, p. 47, 1911.

36. Hunt, Ramsay: The Chronic Progressive Softening of the Brain; Report of Cases with Autopsies Simulating Cerebral Tumor, *New York Neurol Soc.*, May 1, 1906; *J. Nerv. & Ment. Dis.* **33**:779, 1906.

37. Weber and Schultz: Fälle v. Pseudotumor cerebri mit anatomischer Untersuchung, *Monatschr. f. Psychiat. u. Neurol.* **23**:212, 1908, Supplement.

38. Finkelnburg and Eschbaum: Zur Kenntnis des sogenannten Pseudotumor cerebri mit anatomischen Befunde, *Deutsch. Ztschr. f. Nervenhe.* **38**:35, 1909.

In one case of Nonne, the choked disk has followed in three weeks by total blindness; in others (Riggs and Engelhardt) it led to amaurosis in a few months.

In three cases the choked disk disappeared and the fundus recovered normal aspect; in the cases of Rosenfeld the choked disk healed within two months, leaving no trace. In the case of Hoppe, the choked disk disappeared, in the first attack, within six months; the patient had a recurring choked disk seven years later, which disappeared a second time within six weeks, with no other treatment than potassium iodid. In the case of Higier³⁹ the fundus twice recovered normal aspect, and the choked disk reappeared for a third time many years later.

(b) Choked disk was absent in eight instances: Vorkastner⁴⁰ and Henneberg,⁴¹ two cases each; one each of Hochhaus,⁴² Bonnhoeffer,⁴³ Ramsay Hunt and Boetticher.⁴⁴

(c) In the remaining four cases fundus examination was apparently not made.

This gives in at least 75 per cent. of the cases, choked disk; whereas true tumor shows it in from 80 to 90 per cent.

2. One case of Nonne was complicated with homonymous hemianopsia; the case of Gowers, with bitemporal hemianopsia.

3. Motility of the eye:

(a) The exterior musculature of the eyeball showed different anomalies. In five cases nystagmus was present (Sicard, Schroeder, Byrom Bramwell, Nonne, Finkelnburg); in two cases ptosis was observed: Rosenfeld and Knauer;⁴⁵ in three cases conjugated deviation of the eyes was present (two cases of Nonne, and one of Bonnhoeffer); eight cases showed diplopia, and this was produced in six cases by paresis of one or both abducens (Bramwell, Kupferberg, Schroeder, Marinesco, Vincent, Sicard).

(b) The interior eye musculature showed: in four cases, loss of pupillary reaction (three cases of Nonne and one of Kupferberg); the

39. Higier: *Neurol. Centralbl.*, 1910.

40. Vorkastner: *Einige praktische Winke in der Behandlung nervöser Erkrankungen*, Berl. klin. Wchnschr., p. 759, 1905.

41. Henneberg: *Fehldiagnose in operativ behandelten Fällen v. Jackson-scher Epilepsie unbekannten Ursprunges*, Charité-Ann., p. 314, 1905.

42. Hochhaus: *Ueber Hirnerkrankungen mit tötlichem Ausgange ohne anatomischen Befund*, Deutsch. med. Wchnschr., p. 1657, 1908.

43. Bonnhoeffer: *Bedeutung der Jackson'schen Epilepsie für die lokalisationistische Diagnose*, Berl. klin. Wchnschr., p. 935, 1906.

44. Boetticher: *Aerztlicher Verein Hamburg*, Deutsch. med. Wchnschr., 1905, pp. 1909 and 1989.

45. Rosenfeld and Knauer: *Stoffwechselstörung in einem Fall v. Pseudotumor cerebri*, *Neurol. Centralbl.*, 1907, p. 636.

pupillary reaction was not altered in the others, which showed: one, miosis (Henneberg); seven, marked mydriasis, and four, anisocoria.

4. One case was complicated by exophthalmia: Alquier;⁴⁶ one case by dyschromatopsia (probably congenital red-green blindness): Devic-Saleh.

5. Five cases showed alterations in the ear; the loss of hearing was bilateral in four cases (two cases of Nonne, one case each of Vincent and Saleh), and unilateral in one (Finkelnburg).

The comparison of these forty-six cases, controlled by necropsy, shows us that choked disk, mydriasis, nystagmus and paralysis of the abducens, are the most constant eye symptoms in pseudotumor cerebri.

B. CASES NOT CONTROLLED BY NECROPSY

Mohamed Saleh has further collected in his thesis thirty-four cases of pseudotumor cerebri which were not confirmed by necropsy but which, after a critical study of the descriptions, he considers as bona fide cases; they all had been under observation for more than two years, after the disease set in. To these cases of Mohamed Saleh we may further add the following as probable cases of pseudotumor cerebri, although not confirmed by necropsy: One case of Oppenheim,⁴⁷ healed within a year and a half after lumbar puncture; three cases of Babinski and Chaillous,⁴⁸ healed after lumbar puncture; one case of Dor and Jaboulay⁴⁹ observed for eight years; one case of J. N. Roy,⁵⁰ healed after lumbar puncture, and finally the case of Alfred J. Horsey.^{50a}

Considering these cases from an ophthalmologic standpoint, we find as a most important fact that all eye symptoms disappeared with the cure of the disease. There were found: nystagmus in seven cases; paralysis of the abducens in seven; keratitis neuroparalytica in one (Finkelstein); paralysis of the oculomotor in three; exophthalmia in one (Finkelnburg); ptosis in one (Nolen).

Ear ailments were noted in various cases; unilateral deafness appeared in a case of Finkelnburg and the deafness developed as a rule one or two years after the disease set in; only in one case of Nonne did deafness appear as first symptom.

46. Alquier: *Sur le diagnostic des tumeurs de la protuberance annulaire*, *Gaz. d. hôp.*, 1911, p. 873.

47. Oppenheim: *Gesell. f. inn. Med.*, Berlin, Nov. 15, 1897.

48. Babinski and Chaillous: *Ponction lombaire dans les nevrites optiques d'origine intracrânienne*, *Ann. d'ocul.*, July, 1907.

49. Dor and Jaboulay: *Gaz. hebd. d. méd et d. chir de Lyon*, 1902, No. 44.

50. Roy, J. N.: *Annales des maladies de l'oreille, du larynx, du nez et du pharynx*, 1912, p. 1.

50a. Horsey: *Case simulating tumor of the brain in which there was present headache, vomiting and optic neuritis; remarks*, *Ophth. Rec.* 6:636, 1897.

Choked disk was found present in thirty cases, and of these, one was unilateral (Finkelnburg). It was absent in five cases, and in one fundus examination apparently was not made. Of these thirty cases with choked disk, two were followed by complete amaurosis: Finkelnburg and one case Merle;⁵¹ eleven healed with defective vision; sixteen cases receded so completely that neither ophthalmoscopic nor visual examination could detect a trace of the former disease. Under these sixteen cases were found: seven of Nonne; four of Finkelnburg and Eschbaum; one each of Nolen, Velter,⁵² Claude and Baudouin,⁵³ and Unger. In the case of Unger the patient had been nearly blind and recovered normal vision after incision of a serous cyst in the cerebellopontile angle. In the case of Roy the patient was completely blind during two days, and had shown a marked bilateral nervous deafness without alterations of the vestibular function (the Bárány test with hot water remained negative). He recovered, within six weeks, his normal vision and hearing.

In all the cases choked disk appeared as the last symptom of the brain tumor syndrome, and likewise always disappeared last. Finkelnburg and Eschbaum observed in three cases recurrences of choked disk; in one case choked disk disappeared within six months; reappeared two years later, and the patient returned again to normal vision within six months. In another case relapse occurred ten years after the first attack; this patient likewise going back to normal. In a third case, the relapse occurred four years after the first attack, normal vision returned within three months, and choked disk reappeared a third time two years later, normal vision returning likewise after this recurrence. In the case of Dor and Jaboulay, all symptoms, including bilateral choked disk, disappeared after lumbar puncture; the patient suffered a relapse seven years later, with reappearing choked disk, which again disappeared after palliative craniectomy had been done.

From the foregoing comparisons, we may conclude that although retrocession of choked disk in genuine brain tumor is an exceedingly rare occurrence, it occurs rather frequently in pseudotumor cerebri, and consequently we may regard spontaneous retrocession of choked disk as a symptom strongly indicating pseudotumor cerebri. There

51. Raymond François Merle: Deux cas de pseudo-tumeurs cérébrales, *Rev. neurol.* 17:1522, 1909.

52. Velter and Chauvet: Deux cas d'hypertension intracranienne sans tumeur cérébrale guéris par la craniectomie décompressive, *Rev. neurol.* 1911, p. 269.

53. Claude and Baudouin: Un cas de pseudotumeur cérébrale, *Rev. neurol.*, 1911, p. 122.

are other articles in the literature describing retrocession of choked disk; namely, those of Gunn, Yamaguchi, de Schweinitz;⁵⁴ Anderson⁵⁵ with three attacks of reappearing choked disk; Jacobsohn;⁵⁶ Gowers⁵⁷ and Park.⁵⁸ I do not know whether, in these cases, necropsy disclosed the presence or absence of a neoplasm. They are strongly suspected of being cases of pseudotumor.

If the choked disk and other eye symptoms disappear, not spontaneously but after lumbar puncture, we are still not certain of the absence of brain tumor. Flatau⁵⁹ saw choked disk recede to normal after four lumbar punctures. The patient had a relapse three months later and died, the necropsy disclosing a gliosarcoma of the cerebellum. Babinski and Chaillous⁶⁰ observed retrocession after seven lumbar punctures had been made. Necropsy two years later proved the presence of a tumor. Other observations of retrocession of choked disk following lumbar puncture have been made in patients suffering from brain tumor symptoms, by Schneyder,⁶¹ Donath,⁶² Fraenkl,⁶³ Schuster.⁶⁴ But all these cases were under observation only for a few months, and it is, therefore, impossible to classify them definitely. I do not know if there exists a reliable observation of a retrocession of choked disk to normal, in real brain tumor, retrocession being observed at least three years.

On the other hand, it should be remembered that lumbar puncture is often without effect on choked disk as well as on the other brain tumor symptoms. Schneyder and Pérol⁶⁵ each observed two cases, in all of which lumbar puncture had no influence. We may, therefore, conclude, with Babinski and Chaillous and with Lapersonne, that negative influence of lumbar puncture as well as of palliative craniectomy, speaks always in favor of real brain tumor.

54. DeSchweinitz. *Arch. Ophth.* 24:252, 1895.

55. Anderson: *Ophth. Rev.*, 1886.

56. Jacobsohn: *Arch. f. Psych.*, 1896.

57. Gowers: *Med. Ophthalmoscopy*.

58. Park: *Ann. of Otol.*, 1895.

59. Flatau: *Ueber die Rückbildung der Stauungspapille bei Hirntumor*, München. med. Wchnschr., 1905, p. 646.

60. Babinski and Chaillous: *Craniectomie décompressive dans les nevrites optiques d'origine intracranienne*, *Ann. d'ocul.*, 1908, p. 55.

61. Schneyder: *La ponction lombaire comme thérapeutique palliative dans les tumeurs de l'encéphale*, Thèse de Bordeaux, 1908.

62. Donath: *Wien. med. Wchnschr.*, 1903, p. 2306.

63. Fraenkel: *Berl. klin. Wchnschr.*, 1895.

64. Schuster: *Ophthal. Klin.*, 1901, p. 60.

65. Pérol: *La céphalée dans les tumeurs de l'encéphale*, Thèse de Paris, 1907.

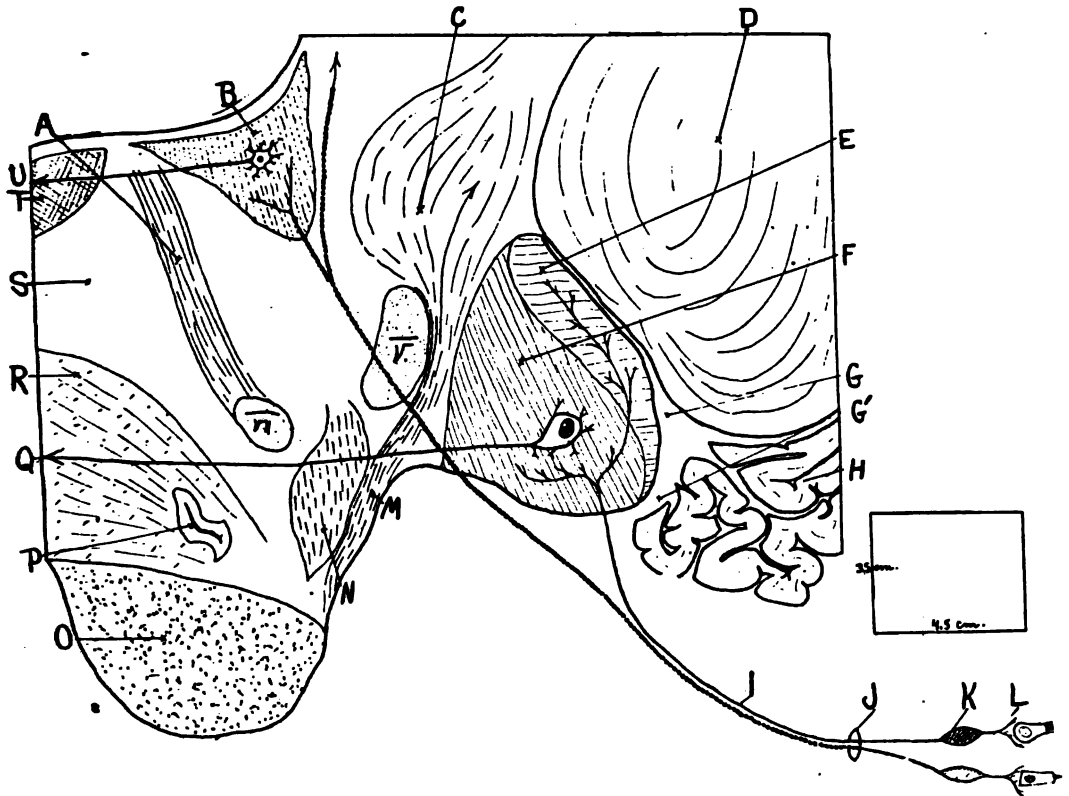


Diagram in illustration of semeiologic references: *A* signifies pars ascendens facialis; *B*, nucleus vestibularis (Deiteri); *C*, corpus restiforme (sensus profundus insciens); *D*, brachium pontis; *E*, tuberculum acusticum; *F*, nucleus cochlearis (sensoric pontine center); *G* and *G'*, angelus cerebellopontinus; *H*, flocculus cerebelli; *I*, nervus acusticus; *J*, porus acusticus internus; *K*, ganglion spirale; *L*, ganglion Scarpa; *M*, fasciculus spinocerebellaris (muscular sense, homolateral); *N*, fasciculus, sensus temperaturae et doloris (heterolateral); *O*, pyramis; *P*, oliva inferior; *Q*, central auditory path passing through the lemniscus lateralis of the other side to the corpus geniculatum internum; *R*, lemniscus sensitivus medialis (sensus conscius superficialis et profundus); *S*, formatio reticularis; *T* and *U*, via vestibularis passing through the opposite longitudinalis pontilis to the bulbar nuclei. The rectangle at the margin indicates the approximate natural size of the section which is 3.5 by 4.5 cm.

PERSONAL OBSERVATION

CASE 2.—Aug. 2, 1916, I saw for the first time the patient, B. L. T., aged 34. She was married for the second time, and stated that her first husband had died six years before from pulmonary tuberculosis. The family physician informed me that he had treated him for syphilis.

She had had by her first husband one child (still living), she had suffered one abortion in the fourth month, and one child had been born dead. By her second husband she had one living child. She stated that for three months she had had swollen glands in the left groin, with fistula; that two months previously she had begun to suffer from headaches which increased in intensity every day. For one month she had had vomiting and vertigo; the vomiting having been so violent that she could no longer eat anything, and the vertigo so excessive that she could not leave home; that it was impossible for her to use street cars as she always had the feeling that the car went up and down like a ship over the waves, and that the houses on both sides were inclined and threatened to fall on her head; that the vertigo had always a longitudinal character, never rotary or circulatory; that her sufferings had been so very intense that she had been forced to stay in bed for several days at a time. Examination revealed: Vision: right eye, 1/0; left eye, 0/5; accommodation, normal. Visual field: There was a very slight restriction of the color fields; but there were no central scotomata. Pupils: The right pupil was narrower than the left. The reaction of both was normal to direct and indirect light on convergence and trigeminus irritation. Fundus: In both eyes there was marked choked disk with hemorrhages; the difference of refraction between border and top of the disk being in the right eye 4 diopters, in the left, 2 diopters. Motility: Constant diplopia in all directions by paresis of both abducens nerves (or contraction of the interni); no nystagmus. Urine: No albumin or sugar was present.

In the right groin a gland developed to the size of an egg; in the left one a fistula was found.

The sensorium was completely free; she had had no fever; there was no Kernig's sign; no rigidity of the neck. Pressure on the mastoid process was very painful, especially on the left side. Headache was localized in the posterior part of the head, in the neck and in the shoulders. She complained of always hearing noises and acute whistles. It was very difficult for her to stand or to walk; therefore a slight astasia existed, but it was not possible to verify a lesion of the pyramids, nor to ascertain whether a lesion of the superficial or profound sensibility was present. One of her neighbors stated that she suffered painful attacks every day, during which she remained quite rigid, could not bend her neck; her gaze would become fixed, sometimes the eyes converged and sometimes the right arm shook.

August 6: Attack of severe pains and involuntary contraction of both arms; the muscles of the breast, neck and shoulders appeared rigid, there was found a slight trismus; she complained of pains in the left side of face and breast; she felt as though her lips and breast were swollen; eye and facial muscles were normal; there was paresthesia in the right arm and in the right leg, and doubtless hypalgesia in the right arm.

August 8: There were paresthesias in both hands and arms; hypesthesias in the left trigeminus field, and bad taste always present in the mouth.

August 10: I had an opportunity of observing an attack which began with paresthesia in both hands; slight nystagmus; trembling in the left forearm and

left hand, and in the right thumb; rigidity of the neck and slight contraction of the left facialis. When standing, she fell toward the right side and backward.

August 15: The pupils were much enlarged. The reaction was normal. There were pains all over the left side of the body, and trembling of the left arm and left leg.

August 16: The blood Wassermann reaction was positive; the patient having the feeling that she might die any moment.

August 20: For two days the patient felt very much better.

August 25: Vomiting disappeared; she had no more diplopia, but headaches continued.

September 1: She had no more paresthesias; she could walk steadily again.

September 8: The patient felt completely normal; she felt well and had gained 19 pounds in weight. The eyes were entirely normal. The vision was 1/0 in both eyes. Choked disk had disappeared without leaving a trace.

During the whole period the patient had no other treatment than a strong dose of iodine.

Aug. 20, 1918: I again saw the patient, two years after the last attack. She stated that one year before (August, 1917), she had had quite a similar attack, but far less severe, for about eight days, with headaches, vertigo and vomiting. She complained that she did not hear so well as before, and especially not with the left ear, and that from time to time she fell toward the left side without being able to explain the reason for falling. Otherwise she felt very well, and had increased 48 pounds in weight. An ophthalmologic examination showed absolutely normal sight for long and short distances. Pupillary reaction, fundus aspect and visual field were absolutely normal. She had no more diplopia. An examination of the ear showed that the function of the acusticus was lost on the left side, without alterations of the function of the vestibularis. The exact report of Prof. Emilio Martinez, to whose courtesy I owe the otologic examinations, was: "The patient has lost the bone conduction in the left ear. The vestibular apparatus is not destroyed, as the patient shows horizontal nystagmus on the turning test." A general examination of the nervous system by Dr. José Valdés Anciano did not show signs of spinal or cerebral disease. It was noteworthy that there were no signs of cerebellar affection. Sensibility and motility were found normal, there was no ataxia, no adiadokokinesis, no asynergia, and no dysmetria. It was on this occasion that Dr. Valdés Anciano induced me to study the interesting thesis of Mohamed Saleh.

September 20: The right pupil was narrower than the left; reactions were normal. Her daughter told me that her memory had begun to fail, and that from time to time she had crying spells without any cause, but that these spells had no spastic character.

Jan. 8, 1919: During recent months she had had no more ictus, but she had had from time to time again slight tremblings over her left hand and left leg, and slight vertigo in longitudinal direction. Combing was very painful for her. In the past four weeks she had lost 10 pounds and felt again severe pains in the left arm and cardiac region with palpitations which lasted sometimes for a few minutes, and at others, for hours. Anisocoria had disappeared, pupils were normal as well as fundi and visual fields.

January 12: The blood Wassermann reaction was strongly positive, in the same manner as two years before. An examination of the blood and of the spinal fluid, which I owe to the courtesy of Dr. Martinez Dominguez and

Dr. Palma, gave the following result: Blood count: red corpuscles, 4,000,000; leukocytes, 11,000; hemoglobin, 70 per cent. There was no hyperglobulia, no abnormal cells and no signs of a destructive process. Spinal fluid: The fluid rushes out under pressure and in a quantity far more than normal. The fluid is clear and does not contain sugar. The total albumin content is 0.7 per thousand. The examination of the fluid revealed:

(a) For syphilis, negative; absolutely negative Wassermann test; (acid butyric test absolutely negative Noguchi-Moor).

(b) For tuberculosis: There was no lymphocytosis and no Fehling's reduction.

(c) For meningitis suppurativa, negative; cultures in Loeffler's serum, blood-agar and glucose-broth remain sterile. Reactions of Pandy, Piralta, Gangui, Violeta and collargol were negative.

There appeared a slight meningeal reaction with marked hyperproduction of the spinal fluid, but without any signs of tuberculosis, syphilis or tumor.

January 24: After a slight quarrel in the family the patient suffered a fresh attack which I had occasion to observe. Sensorium was not lost, although she could not speak. She indicated by nodding her head that she had understood my questions, and executed all the movements I asked of her. She kept the eyes closed; the pupils were of equal size. She could open the mouth only with difficulty. She always lay on her left side, her back curved. Sensibility was diminished on the whole left side, and its transmission was retarded. The tendon reflexes were diminished, the left leg was found in spastic extension and the left arm was found completely paralyzed. The musculature of the face was in constant motion. From time to time she felt intense pains in the left side; the pulse was accelerated to 120 per minute; the neck became stiff, the left leg more extended, the left arm rotated outward and fell with the contracted fist over the left side of the neck and finally over the region of the heart, as if the worst pains were located there. This status lasted about three hours. Later on she felt again quite normal.

April 27, 1919, I saw the patient again. For the past two months she had taken no more potassium iodid. She had been working of late from eight to ten hours a day in preparation for the wedding of her daughter. She felt perfectly well, though she had lost 16 pounds more. She has a good appetite and complains only that from time to time she suffers from vomiting in the morning before she has eaten anything. She has no more headache nor vertigo nor ictus. She suffers rather frequently from palpitation of the heart and states that for the slightest cause or for no cause she bursts out crying. Also her memory has failed somewhat.

An objective examination does not show any signs of tabes. The Argyll Robertson pupil and the Westphal sign are absent. Conscious profound sensibility is normal (diapason). There are no signs of cerebellar affection; no nystagmus can be observed, neither spontaneous nor the form that accompanies voluntary movements; no adiadokocinesis; no tremor and no ataxia. The tendon reflexes are not exaggerated and are on both sides normal. Muscular strength on both sides is good and equal. Her gait is firm and normal. The sensibility is normal and equal on both sides for touch and she localizes well the position of her extremities with closed eyes. The sensation of pain is equal, but perhaps a little under normal on both sides. The sensation of pain is somewhat retarded on the right side of the body (except the face) and does not persist so long as on the left side. The patient contended half an hour after this examination that I certainly must have used the needle with

greater force on her left side than on her right. The sensation of cold (10 C.) is equal on both sides; but she does not feel so well the heat (50 C.) on the right side of the body (except the face). This is especially marked in the right leg, where she confounds almost always hot and cold and where she can distinguish cold only after a remarkably long time.

With eyes closed she shows some unsteadiness of gait. This slight Romberg sign remains the same when she stands on the right leg putting the left heel to the right knee; but she is unable to stand, with closed eyes, on her left leg. The staggering sensation becomes immediately so strong that she falls to the floor, but she can stand well on the left leg with the eyes open.

Her eyes show a slight anisocoria, the right pupil being a little larger than the left, but without any alterations of the pupillary reactions. Fundus and visual examination were normal; there was no diplopia; conjunctival and corneal reflex were present. There were no signs of the trigeminus, facialis or glossopharyngeus being affected. The otologic examination by Prof. Emilio Martinez shows the following conditions:

AD	—	8 m	2.2 m	5/60	0.5 m	9"	8"	12"	O
B	V	V	H	P	CoM	O ^a A	O ^a A	LI	O
AS	o	om	om	o/60	om	o"	o"	o"	o

The vestibular apparatus of both ears was tested by cold water. The left side was completely insensible and revealed no nystagmus. On the right side nystagmus was present, followed by intense vertigo. Conclusion:

Right ear: slight chronic catarrhal deafness (O M C C).

Left ear: complete destruction of acoustic and vestibular functions.

To summarize: The patient shows now abolition of the left acusticus and the left vestibular functions, slight anisocoria without alteration of the pupillary reactions, slight diminution of pain and temperature sensation on the right side of the body and loss of the unconscious muscular sense on the left side of the body.

EPICRISIS

In view of the foregoing described symptoms, we have to consider:

1. *A Malignant Tumor.*—One could willingly believe that the first violent attack was produced by a hemorrhage of one of the vessels of a tumor at the time when it was still very small; but the presence of a malignant tumor (sarcoma or glioma) is improvable because:

(a) From the time standpoint it is not likely that a malignant tumor would produce only such slight symptoms, considering that since the first attack at least three years had elapsed.

(b) There is the contraindication that the patient had gained subsequently forty-eight pounds.

(c) There is a lack of a marked alteration of the blood picture.

2. *A Benign Tumor.*—One might be inclined to believe that the brain became accustomed to a slowly growing benign tumor, but as opposed to this hypothesis we must remember:

(a) That the butyric acid test of the spinal fluid was negative.

(b) That the patient did not show signs of increasing paralysis, but mostly signs of irritation, and that these signs of irritation appeared

at times also on the right side, though they were found mostly on the left.

The best explanation, perhaps, would allow a benign tumor of the left cerebellopontile angle. We know that this originates generally from the sheaths of the acusticus facialis, or trigeminus; that they produce unilateral symptoms and that they act not by infiltration, but by pressure on the region, because they are mostly encapsulated fibrosarcomata or endotheliomata. But against the acceptance of this explanation bear in mind that the patient showed also symptoms of the right side, and that adjacent symptoms were lacking from the trigeminus, abducens and facialis, the symptoms corresponding more to an irregular irritation of the cranial nerves. We must also consider that the glossopharyngeus showed almost no symptoms, nor did the nervus Wrisbergi, and that for some time the vestibular function of the left side was not altered notwithstanding the fact that the function of the acusticus was lost. Brissaud⁶⁶ records an interesting observation which he made of a tumor of the cerebellum which destroyed, by pressure, the radix cochlearis of one side, leaving intact the radix interna (vestibularis). The radix cochlearis is covered and stands in intimate relation to the pia mater. It can therefore easily be destroyed by any process of the arachnoid, while the radix vestibularis is well protected by the corpus restiforme. The fact that our patient did not show cerebellar symptoms cannot be considered as conclusive evidence against the diagnosis of cerebellar tumor, as we know from recent war experiences that unilateral lesions of the cerebellum may be present without symptoms.⁶⁷ But against a cerebellar tumor lies the fact that the patient gained many pounds in weight after the first attack, and that doubtless cerebellar symptoms, especially spontaneous nystagmus, did not develop during the following three years. It remains, therefore, more probable that we are concerned with a cyst of the arachnoid produced by arachnoiditis adhaesiva, which provoked by pressure the irritation of the roots of the various cranial nerves and destroyed the radix cochlearis; provoked the pains, the asthenia, and the falling to the homolateral side by pressure on the left corpus restiforme (bundles passing from the spinal cord and from Deiters' nucleus to the cerebellum). Against the assumption of a tumor of the cerebellopontile angle we must consider the sudden onset of the disease with choked disk; the fact that all symptoms disappeared rapidly, and that disturbance of hearing did not appear until two years after the first attack. As a rule, tumors of the cerebellopontile angle begin with dis-

66. Brissaud: *Le syndrome cérébelleux*, *Maladies nerveuses*, 1:565, 1895.

67. Holmes, Gordon: *The Symptoms of Acute Cerebellar Injuries from Gun-Shot Wounds*, *Brain*, 40:461, 1917.

turbances of the ear, and show only in the later stages of the disease symptoms of intracranial hypertension with choked disk. Regarding our previous experience with pseudotumor, we may state that, as a rule, deafness developed some months after the beginning of the disease.

Furthermore, it is of especial interest to note that the patient presented complete unilateral deafness for more than one year, although the homolateral vestibular function was not abolished. This fact weighs strongly in favor of the assumption that the cause is not a tumor that originated in the sheaths of the acusticus. Henschen found, from a study of 136 cases in the literature, that the tumors of the acusticus originate, as a rule, at the junction of the ramus vestibularis and the acusticus within the porus acusticus internus, which explains why such tumors usually destroy both rami at the same time and why paralysis of the facialis (entering also the porus acusticus internus) follows so soon. There are very few cases reported in the literature showing suppression of the acoustic function without lesion of the vestibular function or vice versa. Eagleton⁶⁸ records that in labyrinthitis serosa the auditory portion of the labyrinth shows greater resistance and that we find, therefore, preservation of hearing after complete loss of the vestibular function. Thomas and Egger⁶⁹ made the same observation in a case of cerebellar tumor. The anatomic investigation showed a complete destruction of the nucleus vestibularis and only slight alterations in the nucleus cochlearis. The somewhat complex topographic relations of the cerebellopontile angle will be borne in mind in the following summary:

SUMMARY

A personal observation of my own shows just the opposite relationship from that of Brissaud, namely, unilateral loss of hearing without loss of vestibular function. This can be explained only by pressure at the edge of the cerebellopontile angle. In the case of Roy there was bilateral nervous incomplete deafness, although an examination of the vestibular apparatus could not detect any abnormality. In his case the deafness was certainly caused by serous meningitis. In our case we might diagnose a cerebellar tumor; but against such diagnosis speaks strongly the lack of spontaneous nystagmus. Eagleton emphasizes the fact that spontaneous nystagmus is apt to be a late manifestation of tumors originating in the cerebellopontile angle, in contradic-

68. Eagleton, Wells P.: The Importance of Aural Symptoms in the Early Diagnosis of Tumor of the Cerebellopontile Angle, *J. A. M. A.* 68:333 (Feb. 3) 1917.

69. Thomas and Egger: *Compt. rend. Soc. de biol.*, 1902, p. 735.

tion to tumors originating in the cerebellopontile angle, in contradiction to tumors growing into it from the cerebellum (involving the cerebellar cortex).

It is noteworthy, furthermore, that the patient showed only slight staggering, with closed eyes, when she stood on both legs or on the right one, but that this staggering became very marked if she tried to stand on the left leg alone, although, with the eyes open, she could stand easily on the left leg. The patient presented, therefore, a unilateral pure Romberg sign through abolition of the homolateral fasciculus spinocerebellaris (unconscious muscular balance). This unilateral Romberg sign is in agreement with the fact that temperature sense and pain sense were slightly diminished on the contralateral side.

The combined symptoms show that an alteration of the meninges in the whole cerebellopotomedullar angle has destroyed the nucleus cochlearis, the radix pontina vestibularis, and the adjacent tractus spinocerebellaris.

The patient has lost her ictus, she now walks firmly and no longer falls down. This fact cannot be explained by the mere statement that the nervous vestibularis is now completely destroyed and that no cause of further irritation exists. To explain it adequately, we must accept the fact that the loss of the vestibular function has been compensated for by a better development of the voluntary muscular sense which accompanies the peripheral sensibility to the cortex, meanwhile the involuntary muscular sense is transmuted by a separate bundle to the cerebellum. This assumption is based on the experiments of Ewald (*Physiologische Untersuchungen über das Endorgan des Nervus octavus*: Wiesbaden, 1892); Ewald cut the nervus vestibularis unilaterally in dogs; after a time normal disequilibrium was completely restored; but by destruction of the sensori motor zone of the cortex Ewald was able to make reappear in their whole intensity all the symptoms of loss of equilibrium in such dogs.

(c) Against the assumption of either a malignant or a benign tumor is the retrocession of the choked disk to normal. Kampherstein saw this in 200 cases only once. It is doubtless an exceedingly rare occurrence in true brain tumor. We have seen before that it occurs quite commonly in pseudotumor. Kornder⁷⁰ has shown that the development of choked disk is dependent on a combination of two factors: increased intracranial pressure and increased venous pressure. It was Graefe's⁷¹ first opinion that choked disk was produced by compression

70. Kornder, L. H.: Hydrocephalus and Choked Disk in Dogs, *Arch. Int. Med.* **23**:197 (Feb.) 1919.

71. Graefe: Ueber Complication von Sehnerventzündung mit Gehirnkrankheiten, *Arch. f. Ophthal.*, Part 2, **7**:58, 1860.

of the sinus cavernosus, causing a stasis in the ophthalmic veins. That this explanation is wrong was proved: (1) By the anatomic investigation of Seseman (1868) who demonstrated the free anastomosis between ophthalmic and facialis veins; (2) by the exceedingly rare occurrence of choked disk in cases of sinus thrombosis, and (3) by the experimental ligature of both venae jugulares (Cushing). On the other hand, Schieck (1910) showed that a mere rise of the intracranial pressure is insufficient to produce choked disk, and that an increase of cerebrospinal fluid is essential. He produced artificial brain tumor by sponge tents under the skull and by the injection of paraffin through the dura; these alone did not cause choked disk. Kornder produced choked disk experimentally in dogs: once by blocking the aquaeductus Sylvii through injection of paraffin, where it appeared on the third day following; another time by intradural injection of liquids under pressure. If, in these cases, he avoided the rise of the venous pressure by the administration of atropin, he prevented the formation of the choked disk. He thinks it therefore probable that an irritation of the inhibitory vagus center produces the rise of the venous pressure followed by an increased secretion of the cerebrospinal fluid. If we apply these experimental observations to our case, we find in the lumbar puncture an increase of spinal fluid. If, however, the choked disk has disappeared, we may conclude that there does not exist a constant rise of the intracranial pressure and consequently no neoplasm.

Jumentié⁷² records in his thesis that hydrocephalus internus adultorum, meningitis serosa, and even tumors of the frontal brain, may produce absolutely the same symptoms as tumors of the cerebello-pontile angle.

3. *A Syphilitic Process.*—Opposed to this conception, these facts may be urged:

(a) The whole clinical feature: though the patient's husband was treated for syphilis, and though her blood Wassermann reaction was found repeatedly strongly positive, she herself had never had symptoms of syphilis during her life. Syphilomata are found, as a rule, at the base of the brain and produce quite another clinical feature. It is not very likely that a gumma would produce the first symptoms of brain tumor, that the symptoms would disappear and that meanwhile the acusticus and the vestibularis would be destroyed.

(b) The examination of the spinal fluid: the negative Wassermann reaction as well as the negative butyric acid test.

(c) The blood count: the lack of hyperglobulia.

72. Jumentié: Les tumeurs de l'angle ponto-cérébelleux, Thèse de Paris, 1911.

4. *Tuberculosis*.—This is excluded by the examination of the spinal fluid: the lack of lymphocytosis as well as the negative reaction with Fehling's solution. Against this supposition we may, furthermore, recall that tuberculomata are tumors chiefly found in childhood. Henschen records that tuberculomata are found extremely seldom among the tumors of the cerebellopontile angle.

5. *Hydrocephalus Internus*.

(a) The cachectic form is excluded by the fact that the patient has gained so much in weight, by the lack of chlorosis, nephritis and polyneuritis, as well as by the lack of insolation, trauma psychicum or physicum.

(b) The chronic, infectious form is excluded by the negative reactions of the spinal fluid, and especially by the fact that the cultures remained sterile.

6. *Meningitis Serosa*.

(a) The acute form is excluded by the lack of somnolence, rigidity of the neck and rise of temperature.

(b) The chronic form is very probable: the persistence of headache, vertigo, ictus, anisocoria and psychic changes show very clearly that there exists in the patient a pathologic process which is still in the process of development. The fact that she presented symptoms of irritation from almost all cranial nerves, from the opticus (choked disk), from the oculomotorius (anisocoria), from the trigeminus (pains over the head and face, trismus), from the abducens (squint and diplopia), from the facialis (twitching of the face muscles), of the acusticus (tinnitus and loss of hearing), of the vestibularis (vertigo in longitudinal direction and general disturbance of the equilibrium), from the vagus (palpitation of the heart and choked disk), from the accessorius (cramps in the neck and in the shoulders), and that the symptoms appeared or disappeared combined or alone, can better be explained by the assumption of transitorily increased spinal fluid in the ventricles and in the arachnoid of the cerebellopontile angle than by the diagnosis of a slowly growing tumor. In favor of this assumption may be urged, furthermore, the increased pressure, the increased quantity and the high degree of clearness of the spinal fluid as it rushes out; also the negative butyric acid test.

CONCLUSION

A woman in the prime of life falls suddenly ill, without any increase of temperature or signs of meningitis infectiosa, presenting all the symptoms of brain tumor: choked disk, headache, vertigo, vomiting. These symptoms, after having appeared most alarming for fourteen days, slowly recede, the choked disk included, so that four weeks later

the patient feels normal. In the subsequent two years a unilateral deafness develops, followed one year later by the unilateral loss of the vestibular function, and from time to time attacks occur which tend to show an alteration in the left cerebellopontile angle. The patient shows now indications of the unilateral Romberg sign, which can be explained by unilateral lesion of the homolateral tractus spinocerebellaris. Pseudotumor can only be diagnosed with certainty by necropsy. If we do not make in this case the diagnosis as benign tumor of the cerebellopontile angle, but incline to that of pseudotumor, it is for the following reasons: sudden onset of the disease; retrocession of all the symptoms within a short time, especially of the choked disk; negative reactions of the cerebrospinal fluid and increase of its quantity; the fact that the unilateral deafness did not appear as a first symptom; the lack of a constant progressive paralysis of the other cranial nerves; the increase of the patient's weight, and finally, the time that has elapsed since the first attack.

I take pleasure in thanking Prof. Dr. José Valdés Anciano for kindly placing his ample library at my disposal.

HYSTERIA IN THE LIGHT OF THE EXPERIENCE OF WAR *

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The war afforded an opportunity such as had never occurred before for investigating hysteria in men. The lessons we have learned can be applied to the elucidation of many of the problems presented by the hysteria of civil life. In this paper I shall attempt to explain the new conception of hysteria which I have been led to adopt as a result of three years of intensive study of the war neuroses.

I begin by proposing a new definition of hysteria:

Hysteria is a condition in which symptoms are present, that have been produced by suggestion and are curable by psychotherapy.

This definition differs from all others in not recognizing an hysterical condition apart from the presence of definite hysterical symptoms. Charcot believed that hysteria manifested itself in two ways: by the symptoms that were obvious to the patient and about which he complained, and by physical and mental stigmata that were present before the obvious symptoms appeared and which persisted after their removal.

PHYSICAL STIGMATA OF HYSTERIA

A characteristic feature of the physical stigmata he described, the chief of which were certain forms of anesthesia and narrow fields of vision, was that the patient never complained about them and did not indeed become aware of their existence until they had been demonstrated to him by the physician. Babinski showed that this was due to the fact that they did not exist until the physician found them, as they were actually produced by him unconsciously in the course of his examination. In a series of 100 consecutive cases of hysteria, which he examined in such a way that no suggestion could occur, neither anesthesia nor a narrow field of vision was ever found.

My investigations, carried on during the past twelve years, entirely confirm Babinski, and we have recently extended our observations still further. A spiral field of vision has often been described as even more characteristic of hysteria than the simple narrowed field described

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by Charcot, which Babinski proved was only the result of the suggestion involved in using a perimeter. When, after the first examination of the field was completed, and second and third examinations were made without interruption, the field was found to become steadily smaller, this being due, it was supposed, to the abnormal fatiguability in such cases. Major J. L. M. Symns and I¹ found, however, that it was simply due to the method commonly used, in which the white disk of the perimeter is brought from without inward at each examination. When it was moved in the opposite direction, an outward spiral instead of an inward spiral field was obtained, the field becoming steadily larger until it reached the normal instead of becoming steadily smaller. By varying the method used, it was possible to obtain an inward spiral with one eye and an outward spiral with the other, or an inward spiral followed by an outward spiral with the same eye.

Accurate measurements made with J. L. M. Symns proved also that the supposed pharyngeal anesthesia of hysteria is a fallacy,² and that the percentage frequency of deficient, average and increased sensibility is exactly the same in patients suffering from hysteria, and even from such hysterical symptoms as aphonia and mutism, as in normal persons.

The areas of cutaneous anesthesia that have been regarded as characteristic of hysteria were shown³ to be simply those areas that an average layman would expect to be anesthetic if he were paralyzed, and which would therefore be likely to develop directly he is questioned on the subject and his cutaneous sensibility is investigated.

MENTAL STIGMATA OF HYSTERIA

Having disposed of the physical stigmata of hysteria, it becomes necessary to examine the mental stigmata. A study of the literature reveals a great diversity of opinion as to their nature. The stigma which is accepted by the largest number of writers, and which is the only one accepted by Babinski, is abnormal suggestibility. But our investigations have proved conclusively that, although abnormal suggestibility renders a man unusually prone to develop hysterical symptoms, there is no one who is so devoid of suggestibility that he may not develop them if the suggestive influence is sufficiently powerful. Whether a given person will develop hysterical symptoms under given conditions depends on the degree of his suggestibility and the strength

1. Hurst, A. F., and Symns, J. L. M.: *Seale Hayne Neurological Studies* 1:15, 1918.

2. Hurst, A. F., and Symns, J. L. M.: *Seale Hayne Neurological Studies* 1:1, 1918.

3. Hurst, A. F.: *Seale Hayne Neurological Studies* 1:7, 1918.

of the suggestion. It is clear therefore that abnormal suggestibility is simply a predisposing factor and is no more a part of hysteria than a tuberculous family history is a part of tuberculosis. Many cases of gross hysterical symptoms occurred in soldiers who had no family or personal history of neuroses and who were perfectly fit until the moment that one of the exceptionally powerful exciting causes, such as occur comparatively rarely apart from war, suggested some hysterical symptom; and after its disappearance as a result of psychotherapy the man was once more perfectly fit, and his subsequent history showed that he remained no more liable than any of his companions to develop new symptoms.

The following experiments made with Major J. L. M. Symns and Dr. R. Gainsborough⁴ show how unexpectedly suggestible the average normal individual is. We examined twenty-eight men who were perfectly healthy and suffering from no kind of nervous disorder, but who were attending an aural out-patient department for deafness, which was either unilateral or more severe in one ear than the other. Both ears were lightly touched, sometimes by ourselves, sometimes by others who did not know we were investigating, and the men were then asked which side they felt more closely. Eighteen replied at once that the ear in which the hearing was less impaired was more sensitive to touch than the other; the hysterical anesthesia which had been suggested by this simple examination was so marked in one case that the man was seen a few minutes afterward putting a pin through the lobe of his ear to amuse his companions. In 1859, Briquet stated that hysterical deafness is associated with anesthesia of the external ear, and this has ever since been regarded as an almost constant phenomenon, which was supposed to help in the differentiation of hysterical from organic deafness. The experiment just described throws light on the origin of this deeply rooted fallacy.

As soon as it is recognized that, although certain mental stigmata predispose to the development of hysteria, they are not themselves a part of hysteria, it becomes obvious that many cases of hysteria will be missed if it is only looked for in so-called hysterical persons. When, on the other hand, it is remembered that there is nobody who may not develop hysteria if the provocation is sufficiently great, it must follow that hysteria is infinitely more widespread than has generally been supposed.

Hysterical Symptoms Produced by Fear.—In the majority of cases very little difficulty is experienced in discovering the nature of the suggestion which gives rise to hysterical symptoms. The chief varieties seen in soldiers may be taken as examples, though each has a much wider application to the hysteria of civil life. In the first place, there are the symptoms which follow the condition of fear. Extreme terror

4. Hurst, A. F., Symns, J. L. M., and Gainsborough, R.: *Seale Hayne Neurological Studies* 1:19, 1918.

gives rise to certain very familiar symptoms, the individual becoming shaky, "paralyzed with fear," and unable to speak—"his tongue cleaves to the roof of his mouth." Under ordinary conditions the cause of fear is momentary and the physical results disappear in a few seconds. But during a heavy bombardment a man often remained terrified for hours. If the tremor, inability to move the legs and speechlessness persisted all this time it was natural that these symptoms of fear, which were not in any way hysterical, should so greatly impress the soldier's mind that the idea of a permanent condition of tremor, paraplegia and mutism suggested itself to him, with the result that when the original emotion had disappeared, its physical expression persisted as hysteria.

In the first two years of the war cases of this kind were given the unfortunate name of "shell-shock" in the belief that they were organic in origin and due to actual concussion caused by the explosion of powerful shells. Consequently, no attempt was made to cure them by psychotherapy, and the treatment by rest and sympathy helped to perpetuate them; this unfortunate result was increased by the use of the word "shell-shock," which gave the patient the idea that he was suffering from some new and terrible disease. When at last the true nature of the condition was recognized, it was found that psychotherapy not only resulted in the immediate disappearance of the symptoms, when they were treated in the special advanced hospitals opened for the purpose by the British and French and later by the Americans, but cases of two and three years' standing were also frequently cured at a single sitting in hospitals in England, such as the Seale Hayne. Although this form of hysteria was most common in neurotic individuals, a large proportion of the patients treated within the first forty-eight hours recovered so completely that they were able to return to the fighting line and showed no tendency to relapse. A few of the patients whose condition had persisted for many months before coming under treatment could not return to France, but such men were always able to go back to their old civil occupation and often had no underlying mental condition requiring further treatment, although in some cases the hysteria was associated with neurasthenia or psychasthenia, or both. Indeed, many patients at once lost such symptoms as headache, depression, insomnia and nightmares, which had troubled them for months or even years, directly the obvious physical symptoms, such as mutism or stammering, tremor and paraplegia, were removed by explanation, persuasion and reeducation.

Hysterical Symptoms Produced by Gassing.—The second great group of hysterical symptoms in soldiers resulted from gassing. The irritation of the eyes, throat and stomach caused conjunctivitis, laryn-

gitis and gastritis, the latter being due to the swallowing of saliva in which the gas was dissolved. The pain caused by the conjunctivitis induced the patient to refrain from opening his eyes with his levator palpebrae superioris; if, however, he tried to open them, his attempt was frustrated by a reflex protective spasm of his orbicularis palpebrarum. Under ordinary conditions the conjunctivitis had improved sufficiently at the end of three weeks for the eyes to be opened without difficulty, but if the patient was led to fear for his vision on account of previous weakness of the eyes, the previous loss of one eye, as in two of our cases, or too prolonged treatment with local applications, bandages, dark spectacles or eye-shades, the voluntary inhibition of the levator might be perpetuated as hysterical ptosis and the reflex spasm of the orbicularis as hysterical blepharospasm. As the uneducated layman associates the idea of blindness with inability to open the eyes many of these patients thought they were blind. Consequently, when they were taught to open their eyes it was found that they could only see indistinctly, as they had hysterical paralysis of accommodation, or less frequently they could not see at all, as they had become so convinced that they were blind that they had ceased to look, and, not looking, they could not see. Simple explanation followed by reeducation in looking resulted in permanent recovery. Similar cases, generally of much less severity and often consisting of nothing more than frequent blinking, are not uncommon in civil life.

In the same way the whispering in cases of laryngitis, which was originally in part voluntary to avoid pain and in part due to a protective reflex, was frequently perpetuated as hysterical aphonia. This was most commonly the case when an expert laryngoscopic examination had revealed the presence of some abnormal congestion or secretion, which led to intralaryngeal medication, as both the diagnosis and treatment afforded the necessary suggestion to perpetuate the idea in the patient's mind that his voice was permanently lost. When these patients were taken away from their unfavorable surroundings and treated by explanation, persuasion and reeducation, without any recourse to suggestion by electricity, anesthetics or other means, they invariably recovered. A series of 100 patients treated at the Seale Hayne Hospital were cured at a single sitting, although the average duration of the aphonia before admission was 205 days. About one third of these cases were not caused by gassing, but by the same suggestive influences as those that give rise to hysterical aphonia in civil life. We believe that the liability to relapse is greatly reduced by our simple method of treatment and the avoidance of suggestion.

The gastritis caused by gassing resulted in vomiting — a protective reflex which fulfilled its object by removing the irritant from the

stomach. The actual gastritis rapidly disappeared, and whenever the vomiting persisted for more than three or four weeks it was always hysterical. A very large number of soldiers were invalided from the service for so-called gastritis, the only symptom of which was vomiting. We found that cases of this sort could invariably be cured by a single conversation, if this was continued until the patient was obviously quite convinced that he was no longer suffering from gastritis and that he could eat anything without fear of vomiting, even if he had vomited after every meal for many months and had been kept on a strictly fluid diet.

Hysterical vomiting is much more common in civil life than is generally supposed. I believe that all cases of the so-called pernicious vomiting of pregnancy are really due to the hysterical perpetuation of the vomiting, which is physiologic during the first few weeks. Similarly the persistent vomiting, which is a common symptom in young anemic women and was formerly regarded as evidence of gastric ulcer, is generally hysterical and is due, like the vomiting in soldiers after gassing, to the perpetuation of a symptom produced originally by an acute attack of gastritis. The vomiting in chronic appendicitis, which may continue even after the removal of the diseased appendix, and of phthisis, and the cyclic vomiting in children, are in great part hysterical. All these varieties of hysterical vomiting can be cured by simple explanation, generally at a single sitting, just as in soldiers, and require none of the treatment by isolation, dieting and drugs, which is commonly given, even by those who suspect that there is a nervous element in the condition.

Hysterical Symptoms Produced by Trivial Wounds of Limbs.—Perhaps the most common of the hysterical conditions in soldiers were the paralyses and contractures,⁵ which followed comparatively trivial wounds of the limbs. A great many different forms were observed, and in many cases the paralysis and contracture were associated with marked vasomotor disturbances, including cyanosis or pallor, a pulse of small amplitude, edema and trophic changes in the skin, nails and bones. At the same time the muscles showed a moderate degree of atrophy, accompanied by an increased irritability to mechanical stimulation and certain changes in electrical reactions, which did not, however, amount to the reaction of degeneration. These changes were often most easily observed under a general anesthetic, which did not result in complete relaxation of the spasm until reaching a stage of anesthesia beyond that in which consciousness is first lost. Babinski and Froment experienced considerable difficulty in producing any

5. Hurst, A. F.: Seale Hayne Neurological Studies 1:43 and 244, 1919.

improvement in the paralysis and contracture by psychotherapy. Impressed by this and by the fact that the associated vasomotor and trophic conditions could not possibly be hysterical, as they were obviously neither capable of being produced by suggestion nor cured by psychotherapy, they concluded that the paralysis and contracture were also not hysterical. They revived the old theory of reflex nervous disorders, with which Vulpian and Charcot had sought to explain the muscular atrophy and spasm that often accompany diseases of joints. They ascribed both the muscular symptoms and the associated vasomotor and trophic disturbances to some obscure form of reflex action.

Our experience has led us to believe that there is no foundation for this theory of Babinski and Froment, and that all the cases they describe as reflex are really hysterical. The immobility and spasm may arise as a voluntary or reflex response to pain, or they may be due to localized tetanus, or to the application of splints or bandages, the abnormal posture assumed, the immobility and spasm being perpetuated by autosuggestion after the primary cause has disappeared, to which very often is added the heterosuggestion involved in treatment by electricity and massage when this is not really required. The hysterical paralysis and contracture which result could invariably be prevented by persuasion and reeducation directly the condition of the wound makes active movement permissible.

The hysterical nature of the paralysis and contracture is proved by their rapid cure with psychotherapy, as has also been shown by Roussy and others in France and Colin Russell in Canada. In a series of 100 consecutive cases treated at the Seale Hayne Hospital, the majority, if not all, of which might have been diagnosed as reflex, as each one of the cases shown in the illustrations of Babinski and Froment's book was represented in our series, ninety-six were cured at a single sitting of an average duration of fifty-four minutes, and the remainder were cured in four days, and two cases in two and four weeks, respectively, although the average duration of treatment before admission was eleven months. It is clear, therefore, that the paralysis and contracture are hysterical, as they are caused by suggestion and cured by psychotherapy.

Disuse of a limb, whether caused by organic disease or hysteria, leads to deficient circulation. This by itself is enough to explain the associated vasomotor symptoms, as they are most marked in cold weather and in persons who have always had a feeble peripheral circulation. They disappear temporarily, as Babinski and Froment showed, by artificially increasing the circulation by the application of heat, and permanently, as we have repeatedly observed, by restoring the power of movement by means of psychotherapy.

Deficient circulation gives rise in turn to deficient nutrition, so that the skin and subcutaneous tissues become atrophied, the bones decalcified as shown by the roentgen rays, and the nails thin and brittle. In a striking case, in which some trophic changes had developed as a result of hysterical paralysis and contracture of over a year's duration, and in which the power of movement was restored at a single sitting, the nails subsequently showed a very definite horizontal line separating the opaque, vertically ridged, thin and brittle part, which grew during the period of disuse, from the pink, smooth and otherwise normal part, which began to grow immediately recovery took place.

The changes in mechanical and electrical reactions and in the deep reflexes were also shown by Babinski and Froment to disappear when the circulation was temporarily improved by immersion in hot water, and we found that immediate and permanent restoration followed recovery from the paralysis and spasm as a result of psychotherapy. These changes therefore are nothing more than the functional effects on muscular tissue of deficient circulation.

In the same way the rigidity of the finger joints observed both in cases of organic nerve injury and hysterical paralysis and contracture, which persists under deep anesthesia, has always been regarded as due to adhesions or fibrous contractures, which only gives way under forcible manipulation with sounds of tearing and resulting effusion. This condition is really the result of some coagulative process in the fibrous tissue caused by the accumulation of products of metabolism, which are normally removed by the blood when the circulation is efficient. It is well known that a slight increase of mobility of such joints follows the application of warmth which improves the circulation, and we have found that complete and immediate restoration of mobility followed the return of the natural circulation as a result of the rapid cure of the paralysis and contracture. This must have been due to the removal of waste products permitting the temporarily coagulated fibrous tissue to assume its normal fluid consistence.

It is thus clear that the so-called reflex nervous disorders of Babinski and Froment are really hysterical, and that the associated vasomotor and trophic disorders are caused by the resulting disuse.

Hysterical Symptoms Produced by Injury or Disease of Nervous System.—The last group of cases is, I think, the most important, because it is one which is very common both in soldiers and civilians, though its true nature is comparatively rarely recognized. It consists of symptoms which are primarily organic and due to an injury or disease of the nervous system, but which are eventually in part or com-

pletely hysterical. When the structural changes produced by an injury or acute disease of the nervous system gradually diminish in extent owing to the disappearance of the vascular and other temporary changes which surround the comparatively small area of total destruction, if indeed such an area is present at all, the symptoms caused by the throwing out of action of the parts controlled by the nervous tissues primarily involved should disappear *pari passu*. Just as the physical signs in slowly progressive diseases, such as tabes and disseminated sclerosis, often precede the onset of symptoms, so in these cases the physical signs are generally still present when the functional capacity has returned to normal, and if the lesion does not disappear completely they may remain as permanent evidence of a past organic lesion.

In many cases, however, a man does not realize that his functional capacity is improving. If he has been hemiplegic, he has in the early days made repeated efforts to move his paralyzed limbs but without success, and he finally gives up the attempt and reconciles himself to the idea of permanent hemiplegia. If his physician is too much concerned with the possible dangers of early movement, he will exaggerate the patient's own fears of permanent disability, with the result that the organic hemiplegia is gradually replaced by hysterical hemiplegia instead of slowly disappearing as the organic lesion becomes more and more reduced in extent. A time may eventually arrive when the hemiplegia is entirely hysterical, but, as already pointed out, the physical signs of organic disease, such as extensor plantar reflex, ankle-clonus, exaggerated deep reflexes and lost abdominal reflex, may still be present on the affected side.

A number of additional signs have been described, particularly by Babinski, which depend on the fact that the behavior of the paralyzed muscles in organic hemiplegia differs in various respects from what an average layman would expect, so that a man with hysterical hemiplegia, the exact nature of which must depend on his own conception of how his muscles would behave if they were paralyzed, fails to show these signs. But if the hysterical hemiplegia was suggested by an organic hemiplegia, these signs would be present, as the patient would be trained by his own organic symptoms to maintain them in an unaltered form when they were no longer organic. Thus while the upper part of the face is unaffected, the lower, including the platysma (Babinski's platysma sign) would be paralyzed, and Babinski's pronation sign and the combined flexion of the thigh and pelvis (Babinski's "second sign") would be present. In the same way the characteristic

6. Hurst, A. F., and Symns, J. L. M.: *Seale Hayne Neurological Studies* 1:113, 1919.

posture of the arm and leg in organic hemiplegia would be perpetuated. We should thus be face to face with a case, in which the symptoms had originated as a result of an injury or disease which is known to result in organic hemiplegia, and in which the incontestible physical signs of organic disease, such as the extensor plantar reflex and the other changes in deep and superficial reflexes, as well as the characteristic posture and the accessory signs described by Babinski and others, are present, although the hemiplegia is entirely hysterical. Such cases can only be diagnosed by experimental psychotherapy. If, as occurred in numerous cases under our care, more or less complete recovery takes place—although of course the permanent physical signs of organic hemiplegia persist—it is clear that the paralysis is almost entirely hysterical, although grafted on an organic basis.

The old method of diagnosing between organic and hysterical paralysis thus breaks down, as the physical signs of organic disease do not, as is too often assumed, indicate that the paralysis is entirely organic, but simply that there is an organic element in the case, which may be quite insignificant in proportion to the hysterical. Moreover, it is no help in such cases to consider whether the patient is or is not neurotic, as the large majority have no personal or family history of neuroses, and are in every way normal except for the particular symptom from which they are suffering. No more powerful suggestion of hysterical paralysis could be imagined than organic paralysis, and no abnormal degree of suggestibility is necessary for its development.

We have seen cases of homonymous hemianopia, which is commonly regarded as always organic, persistent headache, amnesia, and epileptiform convulsions develop after head injuries; paraplegia and persistent incontinence of urine, after spinal injuries; paralysis and anesthesia after nerve injuries in the exact distribution of the nerves, the anesthesia even resulting in accidental burns; all of which were primarily organic and showed the characteristic features of symptoms caused by an organic lesion, although the recovery with psychotherapy proved that they were hysterical. In many cases, of course, recovery was incomplete, the proportion of hysterical to organic incapacity depending on the extent of permanent damage done to the nervous tissues.

In the same way we have found that the well-recognized association of hysteria with disseminated sclerosis is even more common than is generally supposed, that an hysterical element is frequent in tabes and may even occur in such a disease as Friedreich's ataxia. A soldier suffering from the latter disease, who had been unable to walk or stand without assistance and had been unable to feed himself or write for several months, improved to such an extent as a result of a week's

psychotherapy that he was able to walk steadily and use his hands for all ordinary purposes, although of course the physical signs remained unaltered and the ultimate prognosis is as hopeless as before.

We believe that the same principle should be applied to all organic disease, whatever part of the body is affected, and that the possibility of an hysterical and therefore removable element should be considered, however normal the mentality of the patient may appear to be. Our experience further shows that the ideal method of treatment in such cases is a rapid one — by explanation in language suited to the intelligence of the individual, combined, when necessary, with persuasion and reeducation.



AUGUST HOCH, M.D.

1868-1919

Obituary

AUGUST HOCH, M.D.

Through the death of August Hoch, THE ARCHIVES OF NEUROLOGY AND PSYCHIATRY has lost an important member of its editorial board. A wide circle of psychiatric workers and friends feel keenly the passing away of this much beloved man. The loss is most keenly felt by the many who were closely acquainted with Hoch's work and what he might have given to us from the treasures of over twenty years of rare clinical observation and study.

A few years ago signs of a familial arteriosclerosis asserted themselves; a severe attack of sciatica in 1916 yielded but slowly. A change to a well adapted existence in California seemed to reestablish a safe balance; even a somewhat tedious infection of the foot during the last winter had cleared up; but shortly after a happy twenty-fifth anniversary of his wedding, the second day of a visit to San Francisco, where he had gone in part with the hope of working in the Medical Library, he was overtaken by a peculiar collapse, with profound renal insufficiency and a rapid development of uremia.

Hoch came to this country in 1887, when 19 years old, to take up the study of medicine at the University of Pennsylvania. He came from a happy family and a genial circle of friends at the gymnasium of Basel. His father, a minister, had for years been superintendent of the City and University Hospital of Basel, Switzerland. Hopes of an academic career must have been deeply implanted in him and may have determined him, together with his friend, Charles E. Simon, to follow Osler to the Johns Hopkins Hospital. He graduated from the University of Maryland. He worked largely with Henry M. Thomas, and translated Hirt's "Textbook of Nervous Diseases." His first paper dealt with hematomyelia. In 1893 he was chosen to become psychologist and pathologist in the extensive scheme of research laboratories planned by Dr. E. Cowles at the McLean Hospital, and he was sent abroad for further preparation. He went first to Strassburg, with Simon Flexner, to work on brain anatomy with Schwalbe, then to Leipsic, where he endeared himself to Külpe and Marbe and Kiesow in Wundt's laboratory, and finally to Heidelberg where he became one of the contributors to Kraepelin's drive into the domain of a medically useful experimental psychology. Hoch's study of the effects of tea and its oils was made on the basis of some far-reaching generalizations by Kraepelin, especially concerning the facilitation of motor reactions. Hoch married Emmy Muench, of Basel, and then returned to start the

twelve years of work at the McLean Hospital, first at Somerville and later in an excellent laboratory at the new hospital at Waverley, Mass. When I met him for the first time, September, 1895, he was living in the village with his wife and his only child, Susie—in a genial home—and gave his whole enthusiasm and interest to a continuation of his psychologic and anatomic studies.

Edward Cowles had taken the superintendency of the McLean Hospital in 1879, after a brief training at the Hartford Retreat and a period of several years as superintendent of the Boston City Hospital. A man fond of speculation, with a decided yearning for progress, stimulated by Stanley Hall, at that time professor of psychology at Johns Hopkins University, he had an ambition to promote research in his well-endowed institution. His Shattuck lecture of 1885 foreshadowed his program of clinical speculation. He hoped to develop his psychiatric conceptions, leaving the clinical work very much on the old plan of having a few mainly administrative assistants, with the emphasis of innovation resting on the introduction of laboratory investigation. Dr. Hoch became the successor of Dr. Noyes, who had not been especially happy in this semi-academic and nevertheless isolated position. The chemical laboratory, destined to be the birthplace of Folin's reputation, soon was added; staff conferences aimed to harmonize the trends of work. The essentially different Worcester plan of organization laid the emphasis on putting the entire clinical staff on something like a research basis, developing the laboratories in the service of this foundation as means in talents and funds became available, but always with a clear appreciation of the importance of a sound and critical setting. Kraepelin's revolution of psychiatry in 1896 and Nissl's joining the Heidelberg Clinic led to Hoch's second period of work with Kraepelin in 1897. He returned in spirit and fact the full-fledged psychiatric leader of the staff, as well as an especially well trained histopathologist, and one deeply interested in putting the ergograph work into the service of clinical problems. Dr. Cowles never made a complete readjustment to the natural result of these developments, so that the clinical publications were retarded; but Hoch's work became more and more the clinical research with a wise perspective regarding the laboratory investigations for which we all admire him. His interest in the ergograph studies did not cease; but a special report on a patient with alternating days of elation and depression may have served as a damper with regard to the pertinence of the reactions to the specific kraepelinian disease-processes when the dominant process proved to be general paresis. In the first volume of the *Psychological Bulletin*, Hoch summed up the net result of that type of psychologic experimentation in its application to psychiatry.

The period of work with Nissl and Kraepelin in 1897 had a double effect on Hoch. He produced his studies on nerve-cell changes of the cortex in a case of acute delirium and a case of delirium tremens (1897) and on the nerve-cell changes in somatic diseases (1898), full of interesting details, in marked contrast to the numerous writings of that day which saw little more than "chromatolysis" in the cortex pathology. At the same time, from here on, the clinical studies began to predominate: the articles on mania and melancholia and manic-depressive insanity in the *Reference Handbook for Medical Sciences*, the unreality-feelings (1905), the studies on drug deliria (1906), the manageable causes of insanity (1909), the problem of toxic-infectious psychoses (1912) and a most important group of contributions to the study of personalities, the constitutional factors in the dementia praecox group, the mental mechanisms in dementia praecox, the relations of personality and psychosis, and the relation of insanity to the psychoneuroses. All these studies contain little gems of keen and yet direct and simple formulations of well observed clinical cases, in wholesome contrast to Kraepelin's method of presentation, which overwhelms the reader with collections of fragments devoid of personal settings. It was this departure in the work of Hoch that made it possible and perhaps imperative for him to pay more and more attention to the rôle of the personality so strongly emphasized by American workers.

In 1905, Hoch was induced to take the position of first assistant physician of the Bloomingdale Hospital at White Plains, N. Y. He had done some teaching at Tufts Medical School, and he became professor of psychiatry at Cornell Medical College at the same time as he was called to the directorship of the Psychiatric Institute of the New York State Hospitals on Ward's Island, in 1909. As a teacher of the Cornell medical students and of the assistant physicians in the State Hospital system Hoch found opportunities for the fullest expression of his spirit of investigation and formulation. A period of study with Bleuler and Jung and von Monakow, in 1908, gave him an intimate familiarity with the structural and psychoanalytical problems, balanced by an unusually keen sense for intensive clinical study of his patients. It was with the greatest regret that his numerous pupils and colleagues saw him depart for California—a loss compensated only by the hope that in his new environment he would be able to bring out more rapidly monographs based on material from the McLean and Bloomingdale hospitals and the Psychiatric Institute.

From his advent at the Psychiatric Institute he took the greatest interest in the further development of the *Bulletin of the New York State Hospitals* into a psychiatric journal of the first order, the *Psy-*

chiatric Bulletin. In California he hoped to found a special journal, but was finally induced to combine his effort with the newly planned ARCHIVES OF NEUROLOGY AND PSYCHIATRY in order not to divide the efforts where union appeared most urgently needed. During the later years in New York he also proved a most helpful contributor to the interests of the social side of psychiatry, partly in connection with the National Committee of Mental Hygiene. The New York Psychiatric Society and the American Psychopathological Association count him among their most active and influential former presidents.

Hoch was not a generalizer. His strong point and first love was that of appreciation of the finer niceties of description and interpretation. He was not a philosopher, but a man with a keen sense for specific features and aspects of cases or problems. A definite cell alteration, a definite rhythm in the plotted results of his ergograph and kindred experiments, a fact such as passivity or specific traits revealed in the personality-study, which he brought out in collaboration with his friend Amsden, reactions like distressed perplexity, or the death and rebirth concept in some of his patients, would absorb his whole-hearted attention, and would tend toward monographic studies. It is to be hoped that the rich material practically ready for publication will be added to the noteworthy array of publications from his pen.

Hoch's personality was somewhat retiring and at the same time most genial. He was capable of the keenest and heartiest enjoyment of friendship and social happiness with his chosen friends and his family. He gained the warmest affection of his patients. He always remained closely attached to his native country, but was a loyal and warm-hearted citizen of the United States, and deeply appreciative of the beauty of the East and the South, and especially of California.

Hoch's work, his friendship and warm-heartedness will leave an enduring impression on all those who were fortunate enough to know him. His contribution to American psychiatry would fully deserve a memorial edition of his publications, together with the works which we hope his friends and co-workers will bring to speedy completion. What a pity that he should not have seen the day when his many friends and admirers might have celebrated with him the publication of his many-sided and well-poised collected works, somewhat as has recently been done to honor Cornelis Winkler on the occasion of the twenty-fifth anniversary of his professorship of neurology in the Universities of Amsterdam and Utrecht.

ADOLF MEYER, M.D., BALTIMORE.

CHARLES ARTHUR MERCIER, M.D.

1852-1919

Dr. C. A. Mercier died at Bournemouth, England, Sept. 2, 1919. It was my fortune to meet him less than six months ago at the home of Sir William Osler in Oxford and a short description of that "great evening," as Osler put it, may be of interest to American neurologists and psychiatrists.

Last June, when on leave from the army, Sir William and myself were left alone one evening at his house and I looked forward to a quiet dinner with him, but a little twinkle in his eye when I mentioned our solitude gave the secret away, and I knew he had something of interest planned.

appearance, saying good-bye to his nurse, a British matron of appearance, saying good-bye to his female nurse, a British matron of uncertain summers, who had come with him and was to return for him later. Scarcely four feet in height, with enormously bowed legs and long arms, a kyphosis so marked that it seemed as if his great head, which was fully twice normal size, was too much for the spine to hold, a gaunt face with heavy eyebrows shading a piercing pair of eyes — such was Dr. Mercier, a marked victim of Paget's disease. He was dressed in evening clothes with an impeccable purple velvet jacket. Over one ear was a sort of telephone receiver to which was attached a long trumpet of flexible hose, his only auditory connection with the outside world. He talked with a clear, silvery voice using particularly pure English and careful articulation. His flow of language was superb, a great mass of witty conversation, interspersed with brilliant epigrams and anecdotes mostly relative to fifty years of British neurology which he had witnessed.

At dinner he was a perfect fountain of knowledge, appearing to know almost everything about neuropsychiatry and every one connected with it for the last two decades. When the fountain threatened to even suggest a slight drought, Sir William would lean over and whisper into his ear trumpet such pregnant themes as "Hughlings Jackson!" or "Johnathan Hutchinson!" and we would be inundated by a perfect deluge of rambling anecdotes, a veritable spring freshet of information. He was never at loss for a word, a brilliant incident or a humorous story. We are necessarily the listeners, but who could not sit and listen by the hour to such a man!

He told us one or two interesting things about his method of work. For a number of years he had been more or less confined to his house in a chair owing to his infirmity. He arose at four in the morning and, after a bit of fruit, wrote or dictated until ten, when he had a small breakfast followed by a period of a few hours of reading or letter writing. After luncheon came a period of exercise, out of doors if possible, followed by three or four hours of writing or dictating until dinner at eight. This period, he told us, was his best time for work. After dinner came another period of work until twelve or one. Sometimes during the day he rolled his own cigarets and made them a little damp with water so that they would smoke longer. He invariably wrote with a cigaret between his lips. Surely such a routine of labor is seldom depicted in these times. Edison, of course, is noted for his long consecutive hours of work. In the past, John Hunter furnishes the classic example. In 1768, he arose at five and worked until nine in his dissecting room. After breakfast he received patients until twelve, made calls until four, dined, slept for an hour and wrote from six until midnight or later.

A complete story of Mercier's life would be a long one, but a few points may be related. Born in rather poor circumstances in Scotland, he first went to sea as a cabin boy, but soon was drawn to London for his medical education where he fell under the spell of the old London Hospital and Hughlings Jackson. Here he received his groundwork in neurology and mental disease, subjects which filled his life to the end. He was especially interested in the legal aspect of mental disease, and some of his strongest books deal with the relation of crime to insanity. Four of his better known books are: "Criminal Responsibility," a "Textbook of Insanity" (1902), one of the first comprehensive views of insanity in its practical aspect, "Psychology, Normal and Morbid," and his last and perhaps greatest work, published in 1918, "Crime and Criminals."

He was a brilliant speaker and reveled in argument. His nimbleness of brain, strong moral nature and unflinching courage, made him a very formidable opponent in any debate. As a correspondent, especially for the *London Times* and British medical journals, he was considered the most brilliant letter writer of the last decade. It is said that "he never wrote a slovenly sentence and never spared himself the most assiduous effort to make his meaning clear and precise to his readers." He had a weakness which sometimes marred his otherwise convincing logic in that he sometimes sacrificed points in debate in a desire to score, and allowed his wit to run away with his judgment.

This weakness, however, is scarcely noticeable in his medical writings. His style was clear, incisive and accurate, for he was almost a purist in the use of the English language.

He died suddenly, actively at work, a victim of a remarkable malady. As Osler says, he "went down as he promised with all the flags flying." With his death psychiatry loses one of its most brilliant and distinguished ornaments.

HENRY VIETS, M.D., NEWTON, MASS.

Abstracts from Current Literature

THE FREUDIAN DOCTRINE OF LAPSES AND ITS FAILINGS. A. A. ROBACK, *Am. J. Psychol.* 30:274 (July) 1919.

This essay is one in which the actual and psychologic center is unconsciously touched within the first paragraphs, which often are to be esteemed as highly as the first dream. Roback states that the lapse, the doing of something else than that intended, as seen by the common person, has in it the notion of portent, a precursor of a fortunate or unfortunate event. To him it is a symbol. To Freud it is a symbol also, not of extraneous origin, but of a hidden motive in the mind of the person "guilty of the slip."

The author, in his contradiction of Freud, is poised also in a general contradiction of the common person's deep psychology, so like to Freud, and the common person and his notions is a far more dangerous opponent than is perhaps the founder of psychanalysis. No one knows this more shrewdly than Freud and no one has more significantly acknowledged his debt here in a tribute to the curious perception by homo sapiens of just enough of his soul that he may disguise it from himself by a projection outward in an imputation of the wrong as resident outside self. We suspect the author, as one of the race, has himself expressed some such notion in utilizing the connotation "guilty" in defining the one who has made the slip. At least one knows that in violently reacting against tradition there may be concealed a real acknowledgement of it.

It is of interest, then, to extract from this essay the conscious rationalizations affecting the matter of lapses, realizing in it that the author is only more shrewd than the rest of us, locating these slips not in terms of portent or hidden motives, but in psychologic happenings or certain habitudes in no wise to be viewed as conditioned from within, but totally from without, and that in no symbolic manner, but one that is objectively mechanistic in its entirety. It is blanket acquittal which he achieves of man. With it is the usual bitterness against Freud, whom he admires "but"—; "his doctrine should receive its coup de grace;" analysts other than Freud are "devotees," "epigones;" a passing allusion to "complex distilleries" entertains one. All of this may introduce the psychologic values or motives of the contribution with the common unconscious background as occupied with the project of motives outward and outside self.

To present his statement, for there is no argument, the slip is ascribed to a set of causes, variously presented under terms of "repetitionary assimilation," as in saying big bills instead of big pills; in the elimination of prefixes, as "un" or "in," where the lapse is located in the unintentional interchange of negative and positive concepts, this grouped under the general notion of "omissions;" in mistakes due to "anticipation" instead of repetition, as in writing something earlier in a phrase than was meant; in errors of "graphic habituation;" in "perseveration" of a letter to a part where it does not belong.

These are the "mechanisms" which, neglected by the freudians, are here restored by the author. Certainly they appear so obvious that one might wonder at the earlier failure of discrimination. Yet in the midst of what we

assume to be the usual "procession of causes" one is even more driven to wonder at a negation of any deeper penetration, until one appreciates a definite refusal on the author's part of any such excursion. This refusal is not limited in its statement to a ridicule of the unconscious motive, but sharply defined in a method which, after examining the word and the sentence preceding the slip; looks into possible associations where, in opposition to Freud, emphasis is laid "on the actual association in the mind between the word intended and the misexpression," a totally objective relating. He denies any resort to the "hidden unconscious forces that are constantly distilling sexual and other complexes." Nor is the urge to exclude the unconscious limited to the matter of lapses; there is expressed a hope for a final coup de grace of Freud, who has far more than the "Psychopathology of Every Day Life" on his record.

In a way this attempt at a total discrediting of the freudian doctrine of unconscious determination might well orient one as to the value of the specific arguments advanced as alternative explanations of the slip or lapse. It has hindered the author in a wider approach to his own theory of mechanism and limited the argument to statements rather than proofs. It has shut off many sources. Jung long ago in his association studies described in outer associations much of what might be included in the varied mechanisms of "anticipatory" or "repetitory" assimilation, "graphic habituation," etc. Kempf in his "Autonomic Personality" presents suggestions of a result of conflict in the matter of occupation of nerve tracts. Sherrington's studies possess a large value while any work at psychoneurotic symptoms might clarify and reorder the writer's concepts.

It was, of course, an unfortunate assumption that, because of the universality of lapses, one might more readily approach and study mechanisms here than in the compulsion or phobia or other psychoneurotic formations. It is possible, indeed, to differentiate groupings of errors, define them and even make laws for them; but to deny the presence of deeper laws, physiologic and psychologic, toward which any of the above indicated lines might lead, is rather intolerable. Worse, it denies any amplification of theory promised in a contribution such as this. Might the mechanisms, defined by the writer, be correlated to more fundamental notions, both were gainers.

This possibility is denied by the attitude as to the unconscious. It is this which has made the argument thin and possible of no larger valuation than is here attempted.

PARKER, New York.

THE ABDERHALDEN REACTION IN THE ENDOGENOUS PSYCHOSES. GOTTFRIED EWALD, Arch. f. Psychiat. 60:248, 1919.

The author found the Abderhalden reaction positive in 80 per cent. of praecox cases. In 51 per cent. the positive reactions were obtained by proteolysis from brain tissue and genital glands; and in 40 per cent. from the combination brain, genital glands and thyroid. Katatonia and hebephrenia showed the strongest reactions. No parallel was found between the ferment content of the blood and the clinical course of the disease. As compared with 80 per cent. positive reactions in praecox, 60 per cent. of manic-depressive cases and 50 per cent. of hysterical and psychopathic cases gave positive reactions. The observation is considered important that in mania the reaction was often obtained from thyroid alone and in melancholia from liver proteolysis. Although the author strongly maintains the specificity of the reaction, he does not believe that it is of practical value in forensic medicine.

The Abderhalden reaction in psychiatry dates from the publication of Fauser, in 1912, in which he maintained that whereas in organic brain disease, such as paresis and in praecox, the reaction was positive, demonstrating organic proteolysis, it was negative in the so-called functional psychoses—manic-depressive insanity, hysteria and psychopathics. Further experience with the test has been productive of quite divergent opinion as to its value. Recently Van Slyke, Vinograd-Villchur and Losee, from the Rockefeller Institute, working with pregnant serums, adopted for the measurement of serum protease the amino nitrogen determination as being both a quantitative and specific test for proteolysis and superior to the general methods in vogue in the Abderhalden test. Practically every human serum, whether from a male, a pregnant or nonpregnant woman, showed protein digestion with placental tissue prepared according to Abderhalden. They accordingly not only deny any specificity for this test but consider it valueless as a diagnostic measure. If, therefore, the test as originally suggested for the determination of pregnancy has not yet been generally accepted as specific in this condition, one is justified in accepting with considerable reserve the conclusion that the reaction is specific in psychiatry when based on the less well understood, organic changes in mental disease.

SCHALLER, San Francisco.

PSYCHOLOGICAL ANALYSIS AND RE-EDUCATION WITH CASE STUDIES: MARGARET J. HAMILTON, *J. Abnorm. Psychol.* 13:324 (February) 1919.

One meets here "applied psychology" among the psychoneuroses. "Psychoses of rebellion," "psychoses of fear," as terms designating the writer's nuclear notion of the epilepsy previously diagnosed in two of the case reports, show a certain probable drift. These appellations, rather odd to the psychiatrist, go with a strong antifreudian protest; the writer's work is "psychological analysis," not psychoanalysis. Not only, however, is the voice the voice of Esau, but other lineaments are reminiscent; there are "complexes, unconscious motivations, strivings, conflicts, complex discharges," all laid in an amorphous matrix, for no clinical framework is to be seen and the case recitals run along the lines of a fair social service worker's report.

The two cases of epilepsy are "cured." This should arrest attention. The results will gain meaning if one may see by what course they have been achieved. This is the core of the contribution. Clark's excellent work on epilepsy has oriented us as to the usefulness of a considerable reeducation as a corollate of psychoanalytic procedure and physiologic interpretation. In Hamilton's cases it is the reeducation solely which really presents. What is perhaps amazing is the shallowness of the proper reductive analytic process, for it is obvious that the so-called "psychological" analysis is full of lacunae.

Here, then, one poises the question as to the results defined in the reports. In psychoanalysis there is now being more sharply defined notions relating to the necessity of resolving the repressed and relived attitudes of the patient at the site of the analysis. The analyst is driven to the need of breaking through these, as they are re-presented to him. In measure as this is achieved so is the further reduction of the unconscious complexes made possible. It is, moreover, apparent that by and in the same measure is the analyst placed in position where the "prospective" trends or sublimations of the individual may be made to move forward. Certain analysts, indeed, take the position that much of the laborious reduction of complexes might be left if, in the relationship between patient and analyst, there came to be gained a relaxing of the ancient inhibitions, relived at the site of analysis, and there to be undone and released.

It seems as though this were the exact function vaguely described in many words by the author as eventuating in her treatment. The function has been rather blindly performed and quite too idealistically; but what is more important, with no expressed appreciation of the hazards included in this venture, for one knows too well that definite dangers to the patient are inherent in this part of the work, calling for the shrewdest estimate of behavior transformations given in clinical outlines. A certain exaltation of this functional relationship between the writer as the teacher and the patient as the pupil would indicate an affect that might quite as likely be of disservice as service. Awareness of this is lacking, a gap which would be less likely were the writer to have scorned less the experience offered by a just psychoanalytic orientation.

But beyond this one is again confronted with the question as to the wisdom of the psychiatrically unoriented worker interfering in a range where the widest concepts are too narrow. The specific contributions of a reeducation type are useful and define their need; in this, however, they locate for themselves their proper inclusion within the hands of the neurologist and psychiatrist.

PARKER, New York.

HYSTERICAL ANESTHESIA; WITH SPECIAL REFERENCE TO THE
HYSTERICAL ELEMENT IN THE SYMPTOMS ARISING FROM
INJURIES TO PERIPHERAL NERVES. A. F. HURST AND S. H.
WILKINSON, *Seale Hayne Neurological Studies* 1:171 (April) 1919.

Hurst and Wilkinson set out to demonstrate that the paralysis, and more particularly the anesthesia following organic peripheral nerve injury, are often perpetuated as hysterical symptoms and may readily be removed by suggestion. Nine interesting cases are presented, including median, ulnar and musculocutaneous palsies and one instance of hysterical flaccid monoplegia (arm), in which the original anesthesia was presumably due to peripheral anemia following six weeks' splinting. In all the cases both the motor disability and the anesthesia yielded promptly to suggestion therapy, thus clearly stamping them as hysterical. Unfortunately the authors were under the disadvantage of seeing their cases late and the organic nature of the original conditions is not always clear. To be sure, there is a history of bullet wounds and mention of one ulnar nerve operation, but the extent of the original nerve lesions is in doubt. If case notes, including the electrical reactions, had been available, the organic side would have been more convincing. On this point, the authors conclude that, "a gunshot wound in the immediate neighborhood of a nerve produces minute changes in its structure which quickly disappear, but, evanescent as these concussion changes are, they are none the less organic and the paralysis and anesthesia they produce are primarily of organic nature." An interesting feature of the article is the attempt to show that hysterical anesthesia developed without heterosuggestion by a second person. Instances are cited in which patients were first made aware of anesthesia by accidental burns. Babinski's contentions regarding the rôle played by suggestion in the production of "pithiatism" are so broadly inclusive, that it is practically impossible even theoretically to exclude all possible sources. With the A. E. F. in France, the reviewer was interested in trying to eliminate the possibility of this kind of suggestion in some quite recent cases. A few hysterics came directly to his hands without the previous intervention of any medical agency, and in occasional instances even first aid had not been rendered. It seemed reasonable to assume that there had at least been no outside sugges-

tion, but even then all accidental sources could not be absolutely denied. Further, it must be remembered that the existence of hysteria does not by any means rule out the possibility of an element of malingering and burns may be consciously self-inflicted. Rosanoff,¹ in a recent article, stresses the concealed illicit, morally untenable motive of the hysteric and indeed regards it as the only factor which actuates hysterical conduct. Hurst and Wilkinson in this and previous studies have made a valuable contribution to our knowledge of hysteria and have suggested an interesting field for further investigation.

STRECKER, Philadelphia.

TREATMENT AND STUDY OF TWELVE NONPARETIC NEURO-SYPHILITICS TREATED BY INTRAVENTRICULAR INJECTIONS OF SALVARSANIZED SERUM. A. L. SKOOG AND KARL A. MENNINGER, *J. Nerv. and Ment. Dis.* 50:114 (August) 1919.

The technic for preparation of serum is practically the same as that for the Swift-Ellis method. The skull is trephined under local anesthesia with the usual aseptic precaution. The site selected is usually the frontal lobe, although other sites may be used. The authors prefer an area near Keen's point, only instead of 3 cm. above the external auditory meatus, they select a point 5 cm. above and 5 cm. posterior. The insertion, angulation and depth of the needle depends on the site, age and dilation of ventricles. Blunt needles are used (the pressure of cerebral fluid is the same as that of spinal fluid). The quantity removed is the amount that flows out spontaneously. The serum is then injected by gravity or syringe about two thirds to the amount removed.

The laboratory findings showed marked changes for the better in two cases, slight improvement in five, no changes in three (three cases; records were incomplete) and two died.

The authors report twelve cases treated by this method. Two showed marked, and six slight, clinical improvement. Subjective symptoms were more marked than objective findings. Two cases showed no improvement. The authors state that none of the cases were worse after injection. They report two deaths in twelve cases.

The work of the authors is very interesting, but the results as obtained are not at all brilliant, for out of twelve cases treated, two died, one as they admit as a direct result from treatment and the other possibly as an indirect result. They claim "rather remarkable improvement" in only two cases.

The types of cases selected for treatment were such as would ordinarily respond to the usual intensive intravenous injections.

DELONG, Philadelphia.

A PSYCHOLOGICAL STUDY OF SOME ALCOHOLICS. L. PIERCE CLARK, *Psychoanal. Rev.* 6:268 (July) 1919.

One may introduce the pragmatic aspect of this study in two paragraphs by the writer: "They (the patients) are for the most part individuals whom I have very carefully studied over a period of years and one may say they illustrate in a measure the end results of treatment;" "Perhaps in the vast majority of cases one may optimistically hope for an arrest of the habit if

1. Rosanoff, A. J.: A Study of Hysteria Based Mainly on Clinical Material Observed in the U. S. Army Hospital for War Neuroses at Plattsburg Barracks, N. Y., *Arch. Neurol. & Psychiat.* 2: 419 (October) 1919.

proper precautions and lessened social demands are made upon these special types of interiors." From these two statements one is borne to a consideration of outcome in the six patients cited.

In Case 1 there is offered the suggestion of an "ultimate and automatic (?) cure," residing in a marriage undertaken eighteen months after coming under a supervisory regimen pursued by the author and directly following the only spree defacing this clear record. Since marriage there are only occasional drinking periods of "a day or two a year," "when under the sympathetic attentions of his wife he grows mellow." In Case 4, after remarriage, and as in Case 1, the wife has taken over his entire care, as "a mother might a large over-grown boy." He no longer drinks, nor further philanders, but now is the head of a large company. These two cases rather stand by themselves and, as the writer states, are "interesting."

Case 2 is that of a woman, toward the close of the observation period separated from her husband, after an attempt to resume relations and following two years of improvement due to treatment; for a year after this separation another period, free from drink, interposed itself, following which appeared an attachment to an elderly woman from which is dated further though more moderate drinking, "a good deal of moral and mental dilapidation" and "with no further effort to live a healthful social life."

In Case 3 the analysis was broken off, due to a resistance developing on the heels of "an explanatory coup" effecting the homosex motive. His wife, "who gives little concern for the complete reformation of her husband," is considered a "lukewarm ally within the family" and as such to be a "serious drawback to any reformation." In Case 5, following analysis and at the end of several years of good health, the patient represented himself to the author not with the depressive episode earlier enclosing his drinking, but with a paranoid trend. The author views this as uncovered by the analysis. While in this trend "he fortunately stopped drinking."

With Case 6, a psychoneurosis, in which drink appeared as a substitute for unconscious desire to become important, the drinking ceased under treatment. These are the terminations, the end results of treatment. If one is to hope for an arrest of the habit, it is well, as the author implies, "optimistically" to hope.

As clinical groups one is made a psychopath, one a constitutional inferior and two "periodic depressives." This is, perhaps, the significant note of the contribution and deserves a larger emphasis. The matter of psychotic background holds sufficient to give one pause in a too radiant contemplation of cure. The case histories, compact and admirable as records, do not entirely satisfy in a detailing of the psychotic movement. The *deus ex machina* is not always best seen when related so largely in terms of complex and mechanism. Nowhere is the "nothing but" less acceptable than in the alcoholic. In the introduction, however, there is advanced the notion, familiar to us through Jung and Jelliffe, of different unconscious levels with different degrees of intoxication "releasing varying levels of strivings and conflicts." This follows on the heels of a number of discrete and more strictly freudian notions.

It is made obvious by the writer that psycho-analysis, though less favorable for the alcoholic than for other cases, is capable of yielding a needed orientation of the problem. The sharpest illumination appears here by defining in this group a psychotic field still considerably obscured by moral prejudice and a too large assumption of singly effective exogenous agencies.

PARKER, New York.

THE PSYCHOLOGIC ASPECT OF FREE-ASSOCIATION. THEODORE SCHROEDER, *Am. J. Psychol.* 30:260 (July) 1919.

The essay deals with symbolic thinking and presents rather acute observations; this quite apart from the emphasis throughout laid on a general psychic determinism, made perhaps to occupy an over-conspicuous place. The data utilized are from a set of free associations, collected during a kind of reverie, and under conditions where any relation to an analyst or, indeed, to analytic procedure, might well be out of the question. The current organic stimuli, imputed by the critics as the other general agent effective in production of associations and dream content aside from analytic suggestibility, referred to above as excluded, were of a nature which could give no clue, "either in objective connections between the words or between the objective things which these words usually symbolize."

He states his problem, thus stripped unusually clear of the two imputed causal factors, by asking, under the "personal psychic contributions" of his collected free associations, what kind of thinking is involved in these as well as seeking here a deeper dynamic determinant than that resident in the causations imputed by the critics.

In thus seeing the words in his free associations as significant of something emotional in his own psyche, he passes on to a consideration of these words, not as symbolizing something solely objective to himself; they symbolize what he refers to as his "subjective symbolization" of objective realities. This subjective symbolization includes his concepts and feelings in being themselves symbolic of objective things. So the words are the symbols of deeper symbols of the objective realities; the symbols which the words symbolize are thus represented in feelings and concepts.

This point is rather important; it gives a kind of coloration to thinking which in some degree appears not only universal, but also presents a view of thinking as a process which conscious logic has largely appeared to deny. It is not words, symbolizing objects, but words symbolizing symbols, as "symbolizing the symbolization of related objectives," and this "symbolization of objectives" is one which encloses feelings and concepts. It is not merely that the symbol represents an unconscious feeling or idea but that this feeling or idea becomes current only with this symbol as its core. The outer wrapping is the word, another symbol; but beneath is the deeper symbol.

The progression and evolution of thought has admittedly gone on with the increased reference for meanings to other objectives; one objective comes to be more sharply and inclusively defined by others. Yet in the procedure of analysis, this is reversed; psychopathology deals with this variety of language and not with the evolved expression. To criticize procedure one ought to speak its tongue or at least understand it. It is this on which Schroeder insists. There is much to amend, to change; this is welcomed. But in doing this one must go back somewhat to *lingua franca* of primitive thinking. The author's persuasive attempt to make this point and to define these necessities has formed a valuable contribution, framed in the discussion of a set of free associations which illustrate his goal.

PARKER, New York.

AN ADMINISTRATIVE IDEAL IN PUBLIC WELFARE WORK. OWEN COPP, *Am. J. of Insan.* 76:1 (July) 1919.

Most storms of criticism directed against the present care of the insane and the demand for consolidation of state departments, Dr. Copp believes, get their driving force from defects in institutional management. He presents a remedy.

The state must divide its activities into coordinate departments, each one small enough to allow an expert to direct it. In a middle-sized state such departments would be public health, mental health, rehabilitation (reformatories, etc.) and social service. Control in each department would lie with three directors, one a full-time expert. All directorates would come together in a general board of supervision, which would establish its own agencies for investigation and publicity, and for cooperative community service.

Reasons that make such a plan good may be overlooked in the first reading of such a compact paper, although the accompanying diagrams are helpful. (1) The control relation exists in activities which can be fairly represented by the expert. No man is asked to control two such diverse problems as the care of the insane and the care of prisoners. (2) The supervisory relation is located in a board which slights none of the coordinate departments, and which can have for its field of work general problems too big for any specialized directorate, such as those between states and those between state and national organizations. (3) The central commission also can furnish consultation to all departments (the good relation of advice without control) in mental and physical health and institutional management. (4) The scheme protects the autonomy of the unit activities and of the highly trained individual without whom systems tend to go to pieces. It is centralization without a dictator. It appeals to the spirit of cooperation, and its force comes from the power of public opinion.

BOND, Philadelphia.

THE MENTAL ATTITUDE OF THE PENSIONER. A. ROBIN, Seale
Hayne *Neurological Studies* 1:222 (April) 1919.

Robin's article is particularly instructive at this time when our country and the other great nations are facing the tremendous difficulties in the way of a practical administration of the pension lists. The author finds that the neuropsychiatric pensioners are classified in one of the following groups: those who are cooperative and anxious to get well; those who subconsciously, and in some degree consciously, withhold cooperation; and finally the undoubted malingerers who deliberately plan to retain their feigned disabilities. Under the present ruling, the patient is apparently able to leave the hospital at his own volition, without thereby changing his pension status, and consequently, the British government in this respect is more or less at the mercy of the clever fraud. In this connection, it is interesting to recall the recommendation of the Neurological Society of Paris: In general, purely hysterical manifestations entitle one to no recompense. Exceptionally, a percentage of disability may be allowed somewhere between zero and 20 per cent., but in no case should the latter figure be exceeded.

STRECKER, Philadelphia.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, April 17, 1919

GEORGE A. WATERMAN, M.D., *President*

CONDITIONED REFLEXES AND PSYCHO-ANALYSIS. Presented by DR. DONALD GREGG.

Unconditioned reflexes are those in which a given stimulus is answered by a response such as might normally be expected. A stimulus artificially associated with an unconditioned reflex produces a conditioned reflex.

Psychoneurotic persons are sensitive both to slight emotional stimuli and to stimuli from many sources. Unfortunately, accompanying such emotional sensitiveness goes a facility to develop conditioned reflexes which may seriously interfere with efficiency and happiness. Psychoneurotics have an abundance of reactions of all sorts. In fact, "being nervous" usually consists of a realization on the part of the patient that stimuli produce excessive or bizarre reactions on him. Does not psychoanalysis mean merely the study of the mechanism of the origin of such reactions? Considered from the point of view of physiology, are not many of the cases that we see analagous to the dog whose saliva flows when the bell rings, though the piece of meat may be long since lacking? Is not psychoanalysis stripped of its glamour and mysticism merely this? It is easy to talk of psychoanalysis, subconscious buried complexes, mental catharsis, etc., etc., but if it is our object to help our patients to understand their mental life, is it not our duty to have catharsis, like charity, begin at home, and to think simply and clearly ourselves and not to float off into deep water, if we can explain most of our cases in terms of conditioned reflexes and the universal impulse of self-preservation?

A CASE OF CEREBRAL HEMORRHAGE. Presented by DR. E. E. SOUTHARD.

Photographs of a brain from a case of possible lethargic encephalitis were shown. These photographs disclosed localized and diffuse hemorrhages. The case was one giving a history of influenza and showed lethargy as a mental symptom. There were to be seen in the region of the hemorrhages many polynuclear leukocytes grouped about the vessels of the meninges. Histologically, two possible processes were to be seen, one hemorrhagic, the other inflammatory.

ENCEPHALITIS LETHARGICA. Presented by DR. C. B. McDONALD.

In this paper eleven cases of encephalitis lethargica were reported. One case was described with much detail. The other cases were grouped and a study made of symptomatology, pathology and etiology. Every one showed some cranial nerve involvement and lethargy of varying intensity; some showed involvement of the pons, bulb, basal ganglion and spinal cord. All of the patients had fever, and seven of them had spinal fluids with a normal variation. The mortality was two out of eleven cases. The lethargy varied, in some patients associated with delirium. There were no convulsions. The cranial nerve involvement in association with the lethargy superficially characterized the disease in every

case. Pathology was discussed from the point of view of pathologic findings of Netter, Marinesco, Von Econimo, Basil and McGrath. All these authors found the same condition and increased vascularity shown by congestion of vessels, small punctate hemorrhages and engorged veins in the absence of meningitis.

Etiology: No definite organism has been obtained. Fever was present in all cases. The nature of this disease was discussed relative to infantile paralysis, meningitis and botulism. The conclusion drawn was that the disease as encountered in America and as shown by these cases agrees most strikingly with the cases of Von Econimo who described and named the disease. It is of unknown origin of an infectious nature, and occurs following epidemics of infection, commonly spoken of as influenza. For practical purposes its clinical signs and symptoms are sufficiently characteristic to justify accepting Von Econimo's establishment of the disease entity, encephalitis lethargica.

DISCUSSION

DR. E. B. LANE asked, "Is the lethargy continuous or intermittent?" He had seen one patient who had had several attacks and who died at the final attack. He had also seen one case of general paresis which the family insisted developed after an attack of influenza.

DR. W. E. PAUL spoke of seeing a case that continued over fifteen weeks, running a temperature of 101 F. steadily for six weeks. He asked as to the duration of the longest case recorded as recovered.

DR. F. J. FARNELL spoke of seeing a large number of different types of infectious encephalitis at the Providence City Hospital. One case showed no symptoms except that he had been asleep for a week. The Wassermann reaction was positive, and under treatment the man improved. A child with so-called chickenpox and lethargy was admitted, and two weeks later showed blindness, later she developed mumps and scarlet fever and a cerebral hemorrhage with hemiplegia.

DR. GEORGE A. WATERMAN thought it was very difficult to determine just what conditions might be called postinfluenzal during the past winter, inasmuch as a large percentage of the population have had an illness called influenza since fall. In addition to the frequency of diagnosis of influenza, is the uncertainty of its correctness, as many physicians have termed every cold an influenza infection. He had seen several severe cases of myelitis, which occurred from one to three weeks after influenza; three of these cases were of the poliomyelitis type.

DR. E. W. TAYLOR doubted Dr. McDonald's justification of the grouping of the cases with lethargy as a separate disease. He had seen ten cases, some of them with marked lethargy. One of the cases cleared very quickly, but relapsed a week later. The patients understand more than they appear to. Some of the cases are possibly associated with infantile paralysis; others seemingly are toxic in origin; some show parkinsonian symptoms. Possibly the cases might better be grouped as cases of encephalitis subdivided into different types according to their most prominent symptoms, as for example the Parkinson, lethargic type, etc.

DR. McDONALD stated that he had seen one case of six weeks' duration, which was the longest continued case that he had seen, but the literature gave one case that lasted nearly three months. He felt that the literature seemed to justify the recognition of a disease entity, but that many cases seen on further study proved not to be true cases of encephalitis lethargica.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY*Regular Meeting, May 15, 1919*GEORGE A. WATERMAN, M.D., *President*

Instead of the usual papers various members of the society who had seen service in France or this country spoke informally of their observations and experiences as follows:

DR. WILLIAM JASON MIXTER: I feel rather ill at ease in talking to a neurologic society about my work with the American Expeditionary Forces because my work was largely nonneurological. I had expected, when I went over, to stick to clinical work, but owing to the exigencies of the service I was ordered to England the last of May, 1918, on account of the shortage of medical officers. I was appointed district surgeon of the Winchester district. This comprised four large rest camps in southern England, a number of aviation camps, two base hospitals and embarkation work at Southampton. Most of my work consisted of planning the development of new projects with two ideas in view: First, the care of the wounded coming to England from the American forces associated with the French in northern France and Belgium, and second, the care of American troops on their way through England to the front. The number of American troops that went through England in 1918, according to figures I saw in January, 1919, was 1,030,000. Most of these went through England in a few days, some in twelve hours and some in a week. These were the men we were dealing with in large measure, and our main problem was to keep them in as good physical condition as possible during that period, brace them up following their trip across the Atlantic and send them to France fit for their ultimate business. One of the first snags encountered was rations. The English ration of bread, cheese and tea for supper did not satisfy the Americans. After some difficulty a change in this order was secured. Then there was the question of caring for the American wounded returning from northern France, who were being spread in hospitals all over England. I arranged to get them more or less together. At the time of the signing of the armistice we had 12,000 beds in England full of wounded, and 5,000 more Americans in British hospitals. By the first of January our aim of 25,000 beds in England was accomplished. In order to have a hospital or district function well there must be close cooperation which can be secured if men are willing to get together occasionally and talk things over and plan just what each should do.

DR. ARTHUR H. RUGGLES: I will talk briefly on two or three types of neuroses which I saw and in which I was interested. Though they are not new to civil practice, I think they emphasize certain points as to etiology and something partially new as to treatment. The type that I want to speak of are the conversion neuroses. I use the term "hysteria" a good deal—"conversion hysteria"—and use "hysteria" rather broadly, covering some cases that probably many of you would consider anxiety neuroses, but I came somewhat under the influence of Colonel Hurst in England, who considers a very great majority of the neuroses as cases of hysteria, and some of the cases I shall describe really are major hysteria. The anxiety states, neuropathies and psychothenics that occurred as a result of conversion states, may or may not come into the general grouping of hysteria. The conversion

hysteria is much more common in the enlisted men than in the officers. Anxiety states are more frequent in the officers on account of the added responsibility. The most enviable position in the army, so far as the wear and tear on the nervous system is concerned, is that of the buck private.

I was for five months in a hospital in Scotland. At that time I do not think my mind was clear on the war neuroses and some of the men that I saw working there did not have the clear cut conception that more experience and opportunity for interchange of views gave them. The French were able to handle their problem at home, and called some of their best men in at the beginning. They had to consider their man power, and I do not believe any nation in the war knew as much about conservation of man power and carried it out to the degree that the French did. A man with a neurosis, just as a man with a flat foot, had to be returned to the front just as soon as possible, and the French were extremely successful in catching their cases early and returning them soon to the fighting line.

One of the cases I wish to report was that of a British officer seen in a British hospital. He was something of a problem for a time because he would not talk frankly. He came to the hospital with a most extraordinary gait and tremor, and when he attempted to walk he went through the most extraordinary contortions. It was not possible to get him to talk and it was supposed that a shell had exploded near him, and that he had subsequently developed the tremor and gait disorder. He resisted all forms of treatment, suggestion, persuasion, rest, exercise or reeducation, and it was only after a long and arduous time that we were able to get the following history: (which shows the personality and not remarkable reaction of that personality to the strain that he went through). He had lived the life of a country gentleman, and had not done much work. On his first hunting trip he killed a rabbit and was horrified at the sight of the animal's blood. After that he continued to ride and hunt but never killed, and could not bear the sight of a dead animal. The war came on and he felt that he should go, but had a conflict as his wife was pregnant and did not want him to leave, and the doctor said that he must not go as he was worried about her condition. He remained at home in a much disturbed state of mind as all his friends were going. After his wife was well he started off, still a good deal disturbed. He went to the front with a definite conflict, got into action and was simply overwhelmed with warfare. He could not bear the sight of a dead person or think of killing any one and could not sleep, thinking of it all. He had frightful dreams and was much disturbed because he felt that he could not do his duty as a soldier. To strengthen himself, he used to walk through the cemeteries and trenches at night looking at and feeling dead bodies. His commanding officer was wounded, and he went to the hospital in the rear to see him. When he got down there away from the noise of the artillery he found the commanding officer comfortable and was much impressed by the situation. He felt that he could not go back and face things at the front again, and determined to commit suicide rather than go back. He had driven down on a motorcycle and going back purposely ran into a tree, but was merely thrown off. Three times after his return he tried to throw himself under a motor lorry, but was merely made unconscious for a minute. When taken to a hospital it was found that his legs would not hold him up and he developed this extraordinary gait and tremor. When he had told us his

history and we explained the mechanism to him and told him that his nervous system would not stand service in France and that he would be put on home service, he recovered.

That, of course, was called shell shock. This is one of the most pernicious terms. It is used to cover everything from delirium tremens to senile dementia. It not only covers all that, but is so much heard of that it has become a bugbear for the soldiers. It is not allowed in the nomenclature as a diagnosis but the corps men continue to use it. These patients make their own diagnoses and tell the doctor on his rounds that they have shell shock. It is only when you tell them that you do not know what that is and would like to know what they mean by it that they begin to see the light a bit. Only today I heard a Canadian nurse say that a great many of the Canadian soldiers married in France who had been married before and gave as an excuse that they had shell shock and forgot that they had been married. In addition, the soldiers and populace rather think shell shock a natural accompaniment of going to war. They also expected to be permanently disabled from it.

The second case is that of a sergeant in the regular army who had served nine years and had a very excellent record. In April, a shell exploded in some lime near him and splashed the lime into his eyes. This caused impairment of vision, enough to send him to the outpatient department to have his eyes washed out every day for a number of weeks. He did not remain in the hospital. He worried over losing his vision. He became interested in a French woman and when about to marry her received orders to move to another town. She felt that he was trying to escape her and brought suit for breach of promise. He was arrested and put in the guard house. He never had had a black mark against his record in nine years. On the ninth day, in the guard house, he developed complete blindness. He was sent to an American hospital for the blind, and there it was discovered that he had hysterical blindness and he was cured in one sitting.

The third case was that of an artillery officer who did well in the artillery. In October he thought he could do more in the flying service, and asked to be transferred. As a child he had what was called "growing pains," and his nurse talked about them a good deal to him. His legs had been the point of least resistance all his life. After being transferred to the flying corps, he did not do well. In the middle of October, he had influenza and with it a great deal of pain in the legs. When well enough to go back to duty he found, on attempting to fly, that his legs would not work. He was much disturbed as he thought he was making a mess of aviation. The armistice came, but he continued his training. He felt that he was of no use in the army, and would probably just kill himself in flying. He developed paralysis in both legs, with a great deal of pain. He was in bed four months and when finally seen was considered a functional case, and was cured in one sitting and restored to duty. He never went back to flying, but was able to rejoin his organization and came home with them.

In treatment the British at first were divided in their methods, treating by suggestion, reeducation and persuasion. The French have treated these cases largely by suggestion; in the early days the suggestion of an electric current and later by combined suggestion and persuasion with very good results. In the later days the British, under the direction of Colonel Hurst, have treated these cases largely by persuasion, going over the mechanism of the

case, the causative factors and the resulting mechanism in the individual, persuading the patient to use the function that is out of order and gradually increasing its use. I think this is a very logical method. The method of treating with electricity I have always thought gives the soldier the feeling that the condition was pretty severe since it necessitated this form of treatment, whereas the method by explanation and persuasion could clear the thing up in his mind and put him in a better position to resist a recurrence.

Colonel Hurst treats the functional cases without even making a careful neurologic examination. I feel that these patients should be examined. Colonel Hurst thinks that the patient has already tired of being examined but I do not think that one more examination would hurt, and it would impress these patients with the fact that you know something of their cases, and are in a position to deal with it. Many of Colonel Hurst's patients have an organic lesion with functional symptoms. He thinks that if you know there is an organic lesion present, your mind is rather convinced that the case is organic and not functional and your mental attitude toward the case will be very much less positive and in that way you will fail to cure the functional element. When he has completed what should be a cure, if there is any residual left, he makes an examination to determine whether it is organic. I think that is going at the matter backwards.

DISCUSSION

DR. STEDMAN then asked Dr. Ruggles how far psychoanalysis was used in these cases in his experience. Dr. Ruggles replied that in most of them there was no time for detailed psychoanalysis although there were evident complexes. Most of the men thought they got as good results by the direct question and answer method.

DR. THOM stated that he was particularly interested in Dr. Ruggles' statement in regard to hysteria in enlisted men as compared to officers. He never saw a case of hysteria in an officer in England, but at the American Base Hospital No. 117 among 3,000 patients, from 8 to 10 per cent. of them officers, he saw as many hysterical conditions in the officers as in the men. The percentage of deafness, aphonia, paralysis and blindness was as common in the officers as the men.

DR. A. WARREN STEARNS told of his experience in the navy. He was sent out to the Pacific Coast to a Naval Training Station to develop a psychiatric department which had previously not existed. A personal interview was determined on as a means of detecting the unfit. Occasionally as many as 500 men a day came in, and so the interview had to be brief. He found it easier to classify the men socially than mentally, and so attempted to detect any social handicap. Those found to be so handicapped were held for further examination.

Five points seemed to be of particular importance in making this initial survey: (1) Appearance; (2) geographical factor; that is, relation between home and place of enlistment, or evidence of wandering; (3) formal education; (4) occupation; (5) general health.

In this way recruits were divided into three groups. Those obviously well, comprising from 80 to 90 per cent.; those obviously unfit, comprising 1 or 2 per cent.; and a doubtful group held for further examination. Eighth grade education was considered usually to rule out mental defect. A high school career usually ruled out epilepsy, abnormal personality and congenital

conditions. Industrial stability was considered incompatible with mental disease. Various slides showing curves and charts were shown on the screen. The history card was as follows:

Name	Date	No.
Age	Civil Condition	
Home	Enlisted	
P. E.	Appearance	
School		
Occupation		
Med. Hist.		
Alc.	Arrests	
Symptoms	Fitness (1, 2, 3)	

The information in regard to industrial history was so classified that it proved of value in selecting men for special work. Group tests were used, a series being standardized, and his experience showed that you could predict future success or failure with considerable accuracy by means of mental tests. Moreover, it was possible to judge in what branch of the service a man was most likely to succeed.

It seemed to him that in classifying men four factors are of fundamental importance: (1) Physical condition; (2) capacity as shown by psychologic tests; (3) formal education; (4) industrial capacity.

From the foregoing it will be seen that each man is graded according to the navy standard on a basis of 1-2-3-4, as follows: (1) Inferior; (2) low average; (3) high average; (4) superior. Educationally: (1) Less than eighth grade; (2) eighth grade graduate; (3) high school students; (4) college. Industrially: (1) Misfits or failures; (2) unskilled; (3) experienced; (4) skilled.

In addition, each occupation has been given a serial number from 1 to 53. This makes it possible to give every man a numerical formula representing his capacity and training. The serial number representing his occupation is put at the right of a decimal point as it denotes a qualitative factor, the others being quantitative. For instance, 444.4 would represent a man of superior intelligence, college education, and highly skilled, his occupation being an attorney; 111.34 would mean inferior intelligence, education less than eighth grade and industrial failure, his work being odd jobs.

This forms a simple index, making it possible to locate and evaluate men easily. Also each number serves as a check on the other as a man with a 4 in his formula must be taken seriously, and a 1 means that he should be suspected of incapacity. As a matter of fact, the formulas are very consistent, it being rare to find both a 1 and a 4 in the same formula.

DR. DOUGLAS A. THOM: I wish to speak briefly of the work that I was sent over to England to do, to make a report of the British pension system. It is not a scientific talk but will give you an idea of the problem with which England has had to deal and with which the United States at present is confronted but to a lesser extent. There were no statistics available from the National Research Committee at the time I went over so that I had to get my information by talking to different men and by going to different hospitals.

The plan followed in England was this: The men were sent back from the front to hospitals in England and it was attempted either to get the man back to service or, if this did not seem possible, out of the army. A vast number

of functional neuroses patients were sent back and discharged uncured who had perfectly curable symptoms. Their papers were then sent to the Pension Office where recommendations were made on the disability. A man with a functional disturbance of both legs, for instance, would be considered a 100 per cent. disability. Then the papers were sent to the Pension Board and the man was asked to appear before this board or, if unable to do so, to be examined by a local physician who decided definitely on the amount of the pension. In that way the members of the Pension Board, who saw the case only from ten to thirty minutes, made the final decision as to the amount of pension to be paid. Consequently in England today there are about 20,000 functional hysterical patients. In 1917, about 75,000 came up before these pension boards, of which there were about a dozen in Great Britain. At first it was attempted to give these men gratuities running from \$75 to \$1,500. A man would spend it and come back to the Pension Board and demand more money, and if he did not get it, arouse a lot of criticism.

One difficulty in dealing with these men was that when once a man was discharged from the army there was no place for him to go as there were at the time of the signing of the armistice less than 400 beds in England for pensioners, and at the end of 1917 there were 50,000 of these pensioned neuroses in England without treatment and with no means of getting treatment. The pensions were inadequate, especially for the men with families, and a great deal of discontent ensued. (In this connection it is very important to bear in mind that it is much easier to treat the soldier as a soldier than as a pensioner. The greatest weapon that we had in dealing with the functional neuroses was the fact that we had some control over them. In the case of the pensioner he can refuse treatment if he wishes, and you cannot enforce it.) As the disability is reduced the pension is cut down proportionately by the pension board before which the pensioner has to appear at regular intervals. Of course, this leads to dissatisfaction.

At present, in England, many patients with hysterical symptoms of three or four years' duration are being treated and cured at one or two sittings. Many others have been wearing braces and supports and using mechanical appliances for two or three years and will not go to the hospitals. There will always be in England a considerable number of these untreated cases.

Of particular interest, I think, are the cases with convulsions. Personally, I have not seen a case of clinically certain epilepsy where convulsions were produced by the war. Undoubtedly, there were some in such a vast army, but they did not happen to come under my observation.

Another interesting observation probably made by many men was the association of hysterical convulsions with epileptic convulsions in the same person. It was quite surprising to find that patients that had given a history of epilepsy with remission would come back to the hospital and have typical hysterical convulsions so that it is to be considered that the therapeutic triumphs of some of the men of this country have been due to the fact that they have been treating hysterical persons for epilepsy in whom there was a large element of hysteria.

In conclusion I would say that:

1. No patient should be discharged from the army with any curable hysterical symptoms.
2. No patient should be discharged from the army as an epileptic until after he has been observed by a competent neurologist.

3. All cases of concussion and shell shock should be treated because all of these cases do well under treatment.

4. The gratuity system proved unsatisfactory in England and should not be used under any circumstances.

5. The neuroses should have special hospitals with staffs of specially trained men.

In this country at present the War Risk Insurance is taking over the care of the soldiers if they are discharged, but such a discharged man can accept or refuse treatment. There is no way at the present time to hold a man sent to an army hospital for the insane.

In England 10 per cent. of all the men mobilized were asking for pensions. In this country a great many are going to ask for pensions.

It might be of interest to state that there were 18,800 cases of insanity in the English forces and of these only 3,000 were sent to institutions. The rest were allowed to be taken home because public sentiment in England forbade sending soldiers to institutions for the insane. Parents came demanding 100 per cent. disability, and took the men home. I trust that in this country when the insane soldiers are returned home that some adequate measures will be taken to care for them and that they will not be allowed to go to their homes simply because parents wish it. There is too much sentiment attached to the soldier at the present time for his own good, and particularly for the good of the community where he will have to reside.

DR. JOHN J. THOMAS: My work while in France was largely with the base hospital as chief of the medical service, but I was also appointed as consultant to other hospitals in the district. Consequently, I saw a number of neurologic cases outside of our own hospital. The American hospital problem in France was different from that of the other nations because we were so far from home. Owing to the shortage of beds in France in proportion to the number of men engaged, it was impossible for us to keep a man long in a hospital, and those that promised to be cases of long duration had to be permanently evacuated to this country. That affected the handling of the neuroses. Colonel Salmon early devised a most excellent method of handling these neuroses, and established Base Hospital No. 117 for their treatment. It was located at Chaumont, back of that part of the line which was expected to be taken over by the American army, but changes in the plans and advance of our lines made it impossible to get all of these cases back to the base hospital. This problem was met by establishing three other hospitals near the front. In this way these functional cases were largely prevented from getting back to the general base hospital, and came under the care of men trained in their treatment. I think a large percentage of these cases were well in a short time. Schwab reports that 65 per cent. were sent back to the line. (The object in military life is to get a man well and back to the line as soon as possible and you have a tremendous leverage in treatment in having men under military control.) The more severe cases came to Base Hospital No. 117, but a great many of these were put back in shape and into the line.

The handling of neuroses in the army is a difficult problem, especially the distinguishing functional from organic symptoms. The French speak a good deal of a concussion syndrome, but most of the neuroses that I saw were pure neuroses, and not cases of purely molecular changes from concussion.

I saw many cases of melancholia which differed a little from what we see here. In a French hospital at Tours I saw some cases demonstrated by the

physician in charge as pseudoparesis, which seemed to be due to the fatigue and exhaustion of the front line. In general, they were somewhat similar to paresis with ideas of grandeur and considerable mental deterioration, but without positive laboratory or physical signs.

Although the work of the psychiatrists may have been very good, many men came to France with mental defects.

When I was in England, in 1915, I saw more wounds of the brain than I did the whole time I was over on my second trip. The reason for this was undoubtedly the use of the steel helmet. Most head injuries did very well. The wounds of the spinal cord were fully as discouraging as those seen in 1915. This was probably due to the high velocity of the bullets used in modern warfare. Many of the patients died. About 30 per cent. of the wounded extremities showed some paralysis and the great difficulty in treating injuries of the peripheral nerves was due to the great distance from home and the lack of beds. As a result, many of these had to be left for secondary operations after they got home with less chance for recovery.

CHICAGO NEUROLOGICAL SOCIETY

Special Meeting, May 27, 1919, Held at U. S. Army General Hospital 28, on Invitation of the Commanding Officer, COL. WILLIAM N. BISPHAM

NERVE REGENERATION AND NERVE SUTURE. Presented by
LIEUT.-COL. DEAN D. LEWIS.

Colonel Lewis said that within two hours after section of a mixed nerve, evidence of the first step in regeneration could be detected. This first step is the formation of delicate protoplasmic bands which probably originate from the nuclei of the neurilemma. These bands spring from both proximal and distal stump but those from the former are more important, being four times as active as the latter. The protoplasmic bands bridge the gap between the ends, when these are not too far apart, and serve as conduits for the neurofibrils which grow out from the proximal stump. These are produced in great number, many times the number needed for a normal nerve, each axis cylinder sending out a host of them in quest of the distal stump.

When a gap has to be bridged by a transplant, an autogenous transplant is best. The neurofibrils do not penetrate a heterogenous transplant but grow down along its surface. Theoretically, the best transplant would be an autotransplant with Wallerian degeneration; that is, a nerve about nine to twelve days after division, but experimentally such a bridge has not been found to be superior to a piece of normal nerve.

A so-called neuroma forms on the proximal stump in about nineteen days and is made up principally of spirals of neurofibrils which turn back and wind about the axis cylinders, and of ovoid pads which form on the end of the neurofibrils. Experimentally, it has been found that injection of the nerve with alcohol prevents the formation of a neuroma.

Colonel Lewis advocated neurolysis instead of nerve suture unless there is undoubted anatomic block in the nerve. In many cases the nerve is injured by the projectile but not divided, and a frequent type of lesion is that in which the nerve is embedded in a dense cicatrix of surrounding tissue which causes physiologic but not anatomic block. These are cases for neurolysis.

Colonel Lewis operated on two patients before the society. The first was one of high explosive wound reaching from the greater trochanter to the gluteal fold with paralysis and wasting of all muscles below the knee, some trophic disturbance, no pain. The nerve was found in the midst of scar tissue and obviously had been injured, as at one point it showed a hard fusiform enlargement or nodule. The nerve was freed from enveloping cicatricial tissue and then at the point of the indurated enlargement the hard and thickened sheath was trimmed off with scissors. After removal of considerable tough tissue the nerve where injured was reduced almost to normal size and consistence. In the opinion of the operator this was a much better procedure than to excise the inch or two of indurated nerve and suture the ends. The nerve was then embedded in or surrounded by muscles and the wound closed. The prognosis was considered to be good.

The next case was one of extensive shell wound at the upper third of the forearm involving both ulnar and median nerve. The former had largely recovered, but there had been no motor or sensory improvement in the distribution of the median nerve. This was exposed at the upper part of the wound and traced downward until a definite proximal stump was reached. The operator then looked for the distal stump which finally was located about 2 inches lower. As there were extensive scars from elbow to wrist, it was borne in on the spectator that for such surgical procedures the operator must not only *know* the anatomy, but must be intimately familiar with all tissues in the region and must be a surgeon of great skill and tenderness. In this case the ends could not be approximated so the cutaneous branch of the radial was dissected out, a piece excised and used as a transplant, being sutured to the trimmed proximal and distal stumps of the divided median. Transplant and stumps were enveloped in muscles and the wound closed. For tubulization of sutured nerves, fat, fascia, corgile membrane, calf's artery, etc., have been used, but Colonel Lewis believes that all hemorrhage having been controlled, muscles make the best envelope. Colonel Lewis said that the advisability of operating on nerves in the forearm, especially in its lower part, was questionable because the disability caused by the lesion is not great and the postoperative pain is apt to be prolonged and severe. Stiles of Edinburgh and some other surgeons do not operate on such cases. Replying to a question, Colonel Lewis said that he was not an advocate of stripping vessels or nerves of their sympathetic network for the relief of causalgia. He thought the injection of 50 per cent. alcohol into the nerve a better procedure. It relieved the pain, and by the time the effects of the injection wore off the causalgia would have recovered.

Colonel Lewis then presented some patients, although none of them had been operated on long enough to permit one to say anything about the outcome, except those in which neurolysis had been performed. He had on the service some cases in which primary suture was performed in July and August of last year, and there was distinct return of motor power with almost complete return of function.

Two cases that he presented had shown very distinct evidences of return of motor power after neurolysis. One case was one of constriction by a fine cicatricial band, following a high explosive wound of the left arm associated with fracture of the humerus. There had been complete physiologic interruption for six months. The cicatricial band was divided and a muscle neurolysis of the nerve performed. The dissection used to expose the nerve

was made along the intermuscular septum, as few muscle fibers as possible being divided in order to avoid hemorrhage. Within ten days after this operation was performed there were distinct evidences of return of motion in the radial extensors of the wrist. The improvement continued and had been rapid.

In the second case, a machine gun bullet perforated the right arm high up. The bullet had evidently passed along the musculospiral nerve. Physiologic interruption of the nerve had existed for over six months. The nerve was exposed on the inner side of the arm high up before it enters the musculospiral groove. The epineurium was found thickened in two places, and the nerve was imbedded in scar tissue. The nerve was dissected out of this and placed in a new bed. Within twelve days after this operation there was a return of motion in some of the extensor muscles.

Neurolysis is of distinct benefit in such cases for it undoubtedly hastens return of function and prevents the disabilities which arise from continued inactivity of paralyzed muscle groups.

In performing neurolysis, muscles which are comparatively healthy should be used to make the new bed. The dissection, therefore, should be made along intermuscular septums so that the hemorrhage will be reduced to a minimum. When dissected free, the nerve involved is placed in the new muscle bed. This type of operation is to be preferred to the use of free fat, cargile membrane or of a calf's artery hardened in formalin. It is the most satisfactory type of operation for this purpose.

When the nerve has been divided, an end to end suture should be performed. While some doubt has been thrown on Stoffel's work dealing with the internal topography of peripheral nerves, there is no doubt that nonaxial rotation of the nerve is to be desired and that an accurate end to end approximation of corresponding funiculi should be attempted. Epineural sutures of fine catgut or silk are used for this purpose. These epineural stitches pass a little deeper than the epineurium and obliterate any space that might exist between the ends of the nerves. When long defects exist, an attempt to secure end to end anastomosis by posture or displacement of the nerve segments is attempted. He had secured an end to end suture of the sciatic, when the defects measured 7.5 cm. by mobilizing the segments of the nerve and flexing the knee joint.

Defects in the ulnar above the elbow joint may be bridged by dissecting the ulnar nerve out of the groove behind the epicondyle, and displacing it anteriorly, and by flexing the forearm. In all cases where possible, an end to end suture should be made.

If the defect cannot be overcome, nerve grafting should be resorted to, using the autocable graft advised by Huber or calve's fetal sciatic nerve, preserved in 50 per cent. alcohol, as suggested by Nageotte.

These last methods give only a small percentage of successes. Tubulization is uncertain and should be used only in those cases where no other methods can be applied. Resection of a bone, such as the humerus, to overcome a defect in the musculospiral should not be employed. Lateral implantation of a nerve into a neighboring nerve, using the sensory part of the nerve to carry the fibers downward, may be used in cases where the defect cannot be overcome by the ordinary methods. This procedure is practically autotransplantation.

METHODS OF EXAMINATION AND SUPPLEMENTARY MUSCLE MOVEMENTS IN PERIPHERAL NERVE LESIONS. Presented by
MAJOR LEWIS J. POLLOCK.

The examination of the motor functions of the patients suffering with lesions of the peripheral nerves was conducted by means of a spring scales and the results noted on a chart showing the imprint of a hand or a foot, in terms of pounds or ounces of pull. This method enabled him definitely to determine the extent of movement of each segment about a joint and called his attention to certain seeming discrepancies which he demonstrated (Fig. 1).



Fig. 1.—Testing the pronator with spring scales method.

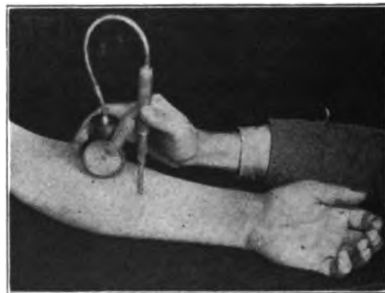


Fig. 2.—Tonometer.

Record of contractures and range of motion was obtained by molding a lead tape about the segments of joints and tracing the outline on paper (Fig. 3).

Imprints of hands and feet were taken and revealed certain characteristic pictures. In many cases the degree of atrophy was shown more clearly by this method than by photographs (Fig. 4).

Tone was measured by ascertaining the millimeters of mercury necessary to insert a blunt plunger a certain distance into a muscle mass. It was found that only for a short time after a peripheral nerve lesion was incurred, was this method practicable. After fibrosis occurred the resistance to pressure occurred as the result of factors other than tone and the method had to be discarded. In the former instance the difference between normal and paralyzed muscles would be as between 180 and 140 mm. of mercury (Fig. 2). Atrophy

was measured by the water displacement method. Contrary to accepted theories, the degree of atrophy did not bear a constant relation to the severity of the lesion. Many cases that were recovering or had recovered showed a great amount of atrophy, whereas cases of complete anatomic division of nerves showed but little. For example, a recovered sciatic nerve lesion showed 500 gm. of loss, whereas a case of a completely severed sciatic nerve with no regeneration showed but 100 gm. of loss. The degree of atrophy is dependent more on the amount of massage, electricity and passive movements the extremity has had than on the severity of the lesions.

The method of sensory examination differs from that ordinarily employed only in the employment of a wisp of cotton saturated with ether to test extreme degrees of cold. This method is more accurate, simple and convenient than the employment of test tubes.

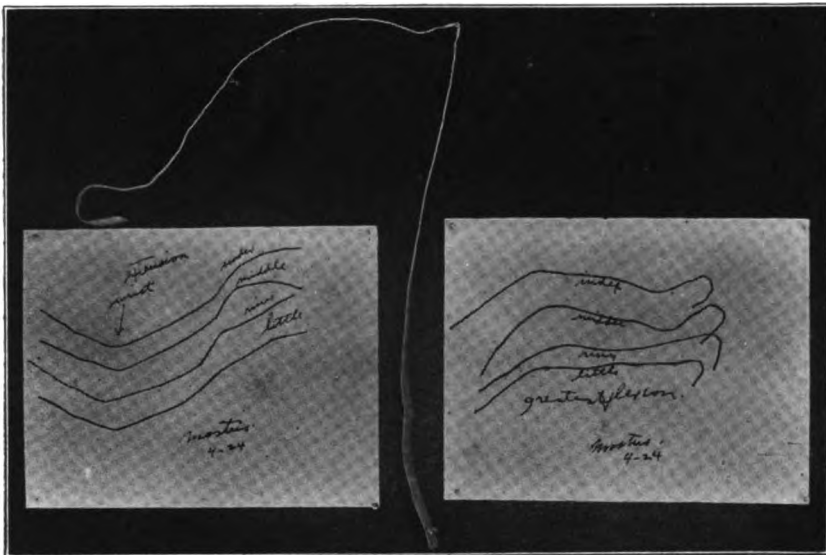


Fig. 3.—Method of recording range of motion.

Major Pollock called attention to the large and constant overlaps of the peripheral nerves for prick pain and showed cases of combined ulnar and median nerve lesions in which the anatomic area of the sensory portion of the median nerve, with the exception of the distal phalanges, was overlapped by the musculospiral and musculocutaneous nerves. This overlap should not be interpreted as a sign of recovery, and return of sense of prick pain in the area of possible overlap would have to be ruled out in studying regeneration.¹

Major Pollock showed about thirty patients to illustrate various "supplementary movements." (Abstract appears as original paper in this issue.)

1. An original paper on this subject will appear in a future number of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY.



Fig. 4.—Imprints of (a) sciatic lesion; (b) normal foot; (c) lower brachial flexus; (d) partial median; (e) ulnar; (f) ulnar; (g) ulnar and median; (h) median; (i) ulnar and median.

CRANIOPLASTY. Presented by MAJOR DALLAS B. PHEMISTER.

Major Phemister performed an operation to illustrate one method of repair of a large cranial defect. On Oct. 5, 1918, the patient had been struck on the head by a bit of high explosive shell which had carried with it a fragment of the soldier's helmet three-quarters inch into the brain in the left parietal region. The patient was not rendered unconscious and had no aphasia, but the right leg and arm were paralyzed. The arm had recovered to a considerable extent, but the leg was still quite paretic. In the last five months the patient had had six jacksonian fits.

The slightly oval cranial defect was about $1\frac{1}{2}$ inches in diameter, and as defects of this size do not grow less, it had been decided to fill it with a bone transplant. The outer table was removed all around the edge of the defect, leaving the inner table. Thus was prepared a narrow shelf or ledge on which would rest the edges of the transplant. This was cut from the outer table of the parietal region of proper size and shape to fit the defect, care being taken to preserve the pericranium. In chiseling this transplant from the inner table it was cracked into several fragments, but the pericranium held them together and the whole was fitted into the defect and secured by suture. Such transplants grow fast to the surrounding bone, retain their vitality, supply adequate protection and, what is not indifferent to the patient, constitute a marked cosmetic improvement.

The American Medical Association will pay 50c each for the April and May, 1919, issues of the *ARCHIVES OF NEUROLOGY AND PSYCHIATRY*. Address to American Medical Association, 535 North Dearborn St., Chicago, Ill.

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ACUTE ASCENDING PARALYSIS AMONG TROOPS PATHOLOGIC FINDINGS *

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NEW YORK

Among the hitherto little known diseases that appeared among the troops in France was a nervous affection of considerable rarity, but of such uniformity in symptomatology and general course that it easily became evident that this was a true disease entity. Gordon Holmes reported a series of cases of this condition in 1917, and later Bradford, Bashford and Wilson,¹ in an excellent article, reported at considerable length on the disease from observations on a series of thirty cases. The comparative rarity of the condition may be judged from this number as it represented all they could assemble from the large number of British troops in France, and it probably represents a goodly proportion of all the cases that occurred.

The writer saw four or five of these cases in eleven months' service with the British Expeditionary Forces, and in nine months' service with the American troops in France, but one case came under his observation. I cannot but feel that this does not represent a fair proportion of the cases that occurred among our troops. Not a single case was seen by me in the big hospital group at Bazoilles, where seven base hospitals had a total of nearly 20,000 beds. The one American patient reported in this article was seen by me in a field hospital to which he had been sent with the diagnosis of psychoneurosis. The divisional psychiatrist, Major George A. Blakeslee, recognizing that the disease was unfamiliar to him, asked to have the writer see the patient. Obviously, the condition was not recognized up the line as being of an organic nature and very likely a number of other similar cases may have slipped through on a similar diagnosis. Lieut.-Col. Sanger Brown, 3rd, told me when I spoke to him of the disease that he was certain

* Read before the Forty-Fifth Annual Meeting of the American Neurological Association, Atlantic City, N. J., June 16-18, 1919.

1. Bradford, Bashford and Wilson: *Acute Infective Polyneuritis*, *Quart. J. Med.* **12**:88 (Oct.) 1918; (Jan.) 1919.

he had seen one case of it at Base Hospital No. 8 at Savanay which had slipped through undiagnosed.

Bradford's careful description of the clinical phenomena of the disease corresponds exactly with that of all the cases observed by the writer. A short fever of from 100 to 103 F., lasting from two to four days, accompanied by severe headache and general pains, subsides and leaves the patient feeling comfortable enough to continue his usually quite arduous duties in the line. In the latent period that now intervenes no symptoms are to be observed for a period of four or five days to a month or six weeks. Following this latent period, the par-

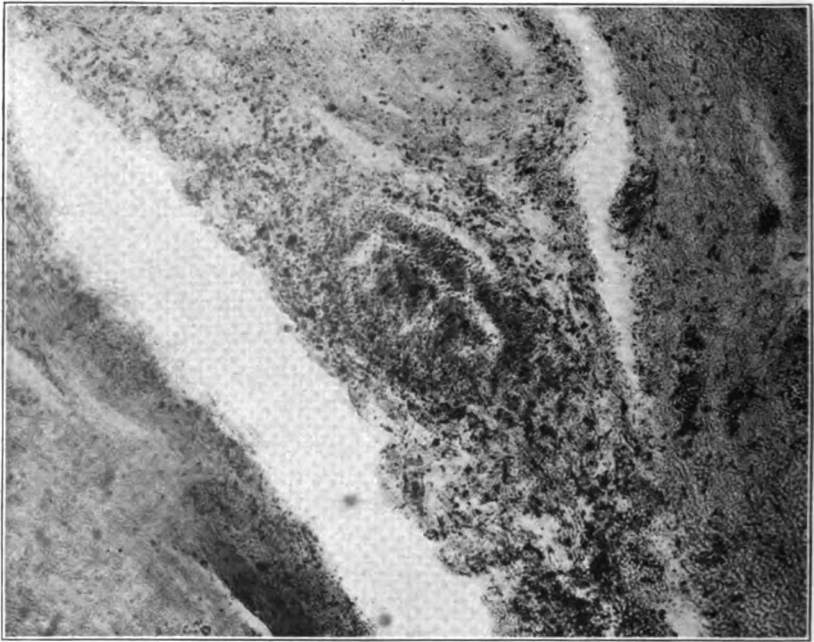


Fig. 1.—The membranes of the cord, low power. Case 1: At the lower left hand corner is the surface of the cord covered by thickened pia. To the right of this is the arachnoid, much thickened, hyperemic, with the tissue spaces filled with red blood cells and detritus. At the right edge of the illustration is the dura, which is adherent to the arachnoid at the lower part.

alytic stage makes its appearance usually quite suddenly. As a rule, the patient suddenly finds his legs getting weak so that he is obliged to sit down. After resting a while he tries to arise and finds that he is unable to do so. When he is removed to the hospital it is found that both legs are completely paralyzed. This paralysis is of an extreme flaccid type, all tendon reflexes being absolutely abolished. At this time the patient is usually found to have a slight fever (99-101 F.),

and frequently complains of pain in the back. The paralysis usually progresses in an ascending fashion. The trunk musculature becomes progressively involved, and after from about 24 to 48 hours a paralysis, similar to that in the legs, is seen in the arms. Following this, the phrenic and vagus nerves may be affected and the patient dies of respiratory paralysis. If this does not occur the paralysis may ascend so far as to involve both facial nerves. Bradford has seen but one case in which the sixth nerve was involved, but never any of those located more cephalad.

When the paralytic stage is fully developed, the patient is practically completely paralyzed from the level of the seventh nerve down. If

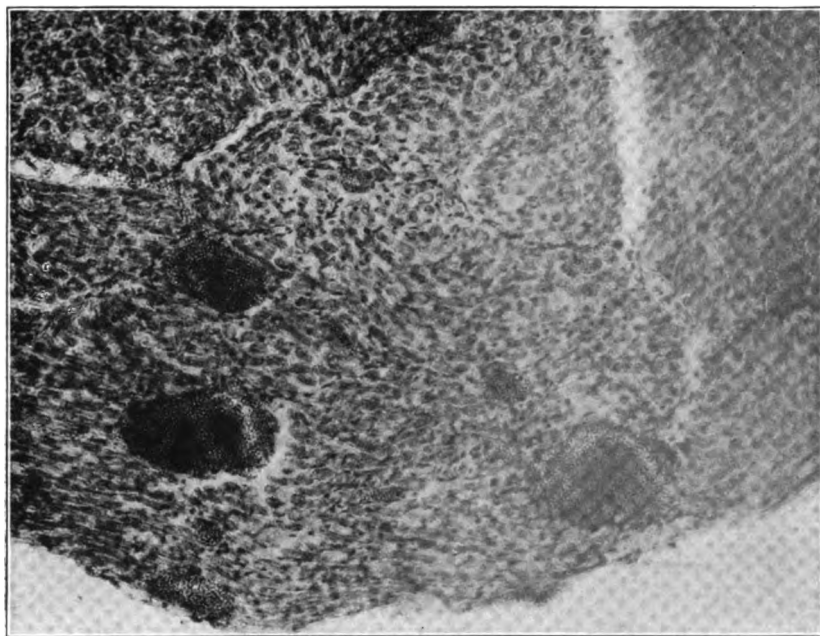


Fig. 2.—Anterior nerve root in the arachnoid. Case 1: showing marked hyperemia and hemorrhage.

any part of the somatic musculature is spared, it is usually the distal parts of the limb. Thus a patient may be able to move his hands fairly well long after motor power is lost at the elbow and shoulder. The paralysis is always of the flaccid type with complete loss of the tendon and superficial reflexes. Sensory loss is always much less marked than motor. In early cases no sensory involvement can be determined. Later in the disease there is present a glove and stocking shaped anesthesia of much the same type as is seen in multiple neuritis. The sensory signs, like the motor, are always bilateral. The prepon-

derance of motor over sensory manifestations is important in view of the pathologic findings. The sphincters are rarely, if ever, affected. The blood may show a moderate leukocytosis. The spinal fluid is always completely negative.

The mortality is fairly high. In Bradford's thirty cases, death occurred in eight, half of the deaths occurring within one week of the onset of the palsy. In the nonfatal cases the paralysis remains stationery for a while, and then starts slowly to improve. Usually the face first shows improvement, and then slowly the arms and legs get stronger, and after from six to eight months the patient has recovered without residual signs of the disease.

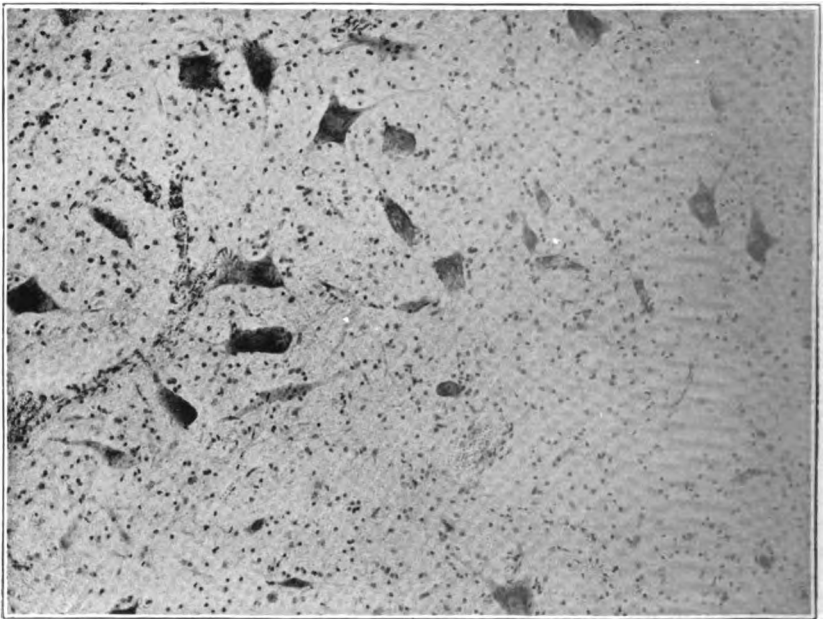


Fig. 3.—Anterior horn, low power. Case 1: showing especially the increase of the neuroglia cells.

PATHOLOGY

Bradford in the article mentioned in the foregoing, gives his results from a study of the postmortem material from six cases. Unfortunately, he makes very little mention of the condition of the meninges in which the most striking changes were seen in my cases. In one case only does he mention the membranes (Case 2), and then observed macroscopically "great edema of the pia-arachnoid and marked distention of the theca." In the spinal cord itself he noted a number of changes in the nerve cells, especially those of the anterior horn. In

some of his cases these cells were more severally affected than in others. In the more advanced cases the motor cells showed eccentricity of the nucleus, finely divided tigroid substance, which was grouped around the periphery of some cells and gathered in the center of others. These, which he shows in excellent illustration, present quite the typical central chromatolysis of secondary degeneration. Many of the changed cells that he describes and pictures, especially the shrunken ones with crenated outlines, appear more typical of post-mortem changes than any known type of cell degeneration. Likewise, I am inclined to doubt the pathognomonic significance of the deep diffuse staining of certain tract cells described by Bashford, for such cells

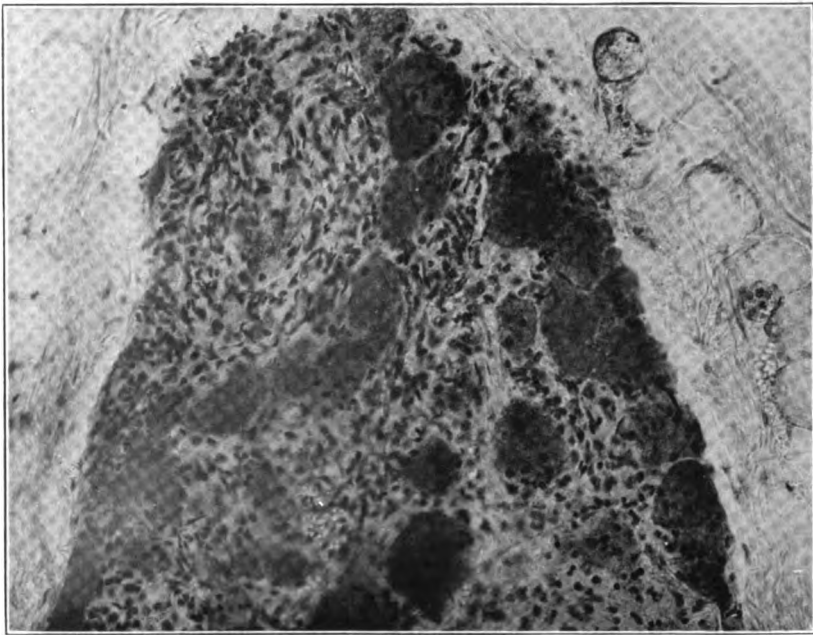


Fig. 4.—Posterior nerve root ganglion. Case 1: showing the increase of interstitial cells and diffuse staining of the nerve cells.

are frequently seen in control cords where no nervous disease had existed. Axis cylinders sometimes appear swollen. He describes some cells in which the outlines were definitely hollowed out where a round cell was applied. This is certainly neuronophagy and must be considered a true part of the pathologic picture.

Besides the changes in the nerve cells, Bashford describes a marked increase in the number of "small round cells" in the gray matter of the cord. This he describes "a round cell infiltration," but he makes a sharp distinction between this and the infiltration of the vessel

sheaths with lymphocytes seen in poliomyelitis. From Bashford's description and pictures, as well as from the study of my own sections, it is evident that these cells are small, young neuroglia cells, and not cells of inflammatory mesodermal origin. In the white matter of the cord, Bashford describes little that is pathologic except evidences of Wallerian degeneration in some of the fibers and an increased number of small, round cells (neuroglia) in the region of the anterior and posterior roots.

The posterior root ganglions of four of Bashford's cases appeared negative except for some increase in the number of small round cells.

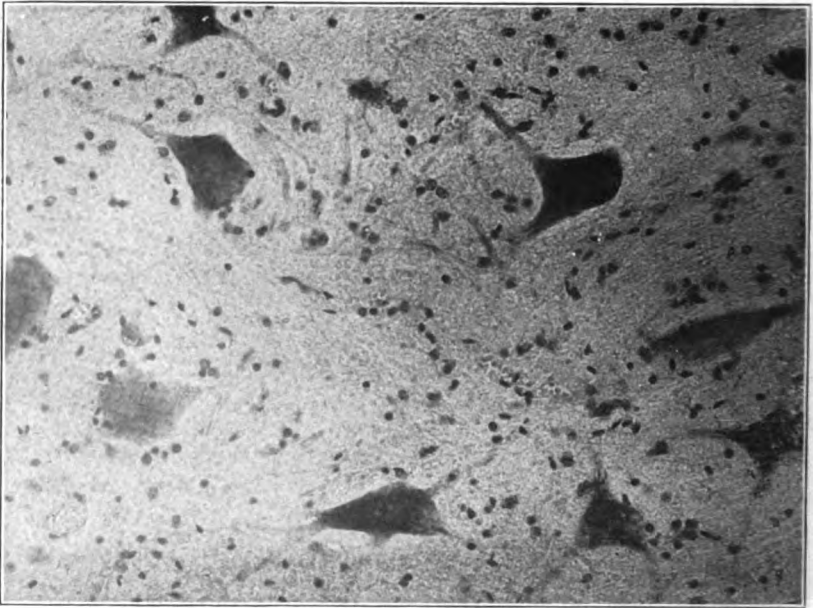


Fig. 5.—Anterior horn, high power. Case 1: showing increase of neuroglia and diffuse staining of nerve cells (acute cloudy swelling).

In one case the cervical ganglions appeared normal, while those from the lumbar region were markedly altered. These showed eccentricity of the nucleus, disappearance of the tigroid and vacuolation. The number of "round cells" was increased.

The peripheral nerves examined showed similar features of varying intensity in different parts of the same nerve. In some cases a partial Wallerian secondary degeneration was seen, and in others an active neuritis with hyperplasia of the Schwann cells, inflammatory exudate of "round cells," and hemorrhages. Sometimes the medullary

sheaths were quite swollen and the axis cylinders were sometimes swollen, irregularly stained and fragmented.

The cerebral cortex and the cerebellum showed the same increase of neuroglia cells as was observed in the cord.

Muscles showed signs of fatty degeneration with osmic and Marchi stains.

Bashford was able to produce the disease in monkeys (*Macaccus rhesus*) by intradural injection of a glycerin emulsion of the spinal cord of fatal human cases. The clinical features and pathologic findings in these monkeys were the same as in man. Likewise the disease was similarly transmitted from monkey to monkey.

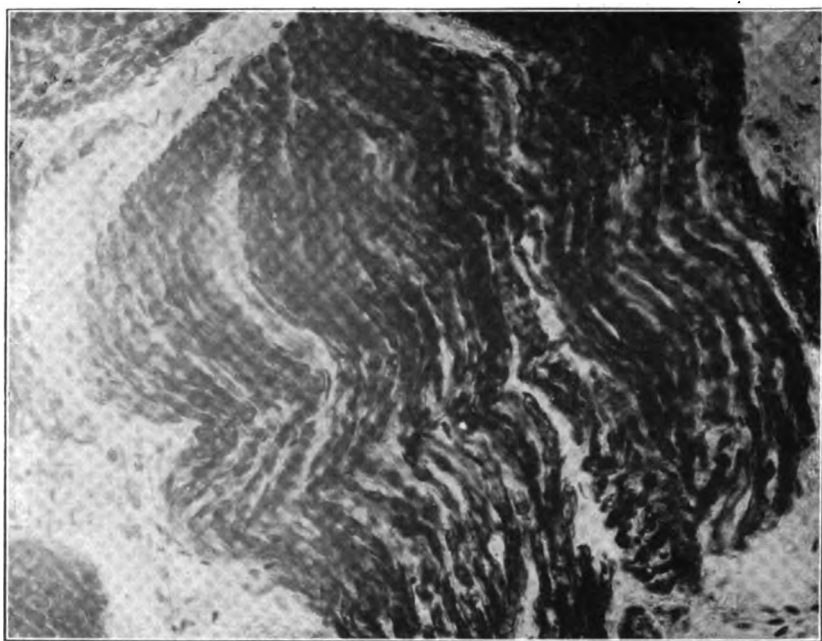


Fig. 6.—Anterior nerve roots in the arachnoid, high power. Case 1: showing the beady appearance of the axons in acute degeneration.

Wilson has made a careful study of the bacteriology of the disease and succeeded in isolating an organism in pure culture that produced the disease in monkeys and which was recovered in pure culture from the inoculated monkey after death. He describes the organisms as being "minute rounded, oval or kidney-shaped bodies, measuring 0.2 to 0.5 μ in diameter, occurring in groups, more rarely in pairs or short chains." In young cultures it appears as a darkly stained spot, eccentrically placed, surrounded by a narrow faintly stained area. In

older cultures the organism swells and loses its selective staining. It is anaerobic and grows only on Flexner and Noguchi's tissue agar-serum bouillon medium.

AUTHOR'S CASES

The material for this paper consists of the spinal cords from two cases. Only the first case was seen by the author before death. Neither history is available at present. The history and other records of Case 1 are in the possession of Major Blakeslee, who has not yet returned from France. The history of Case 2 was transmitted verbally

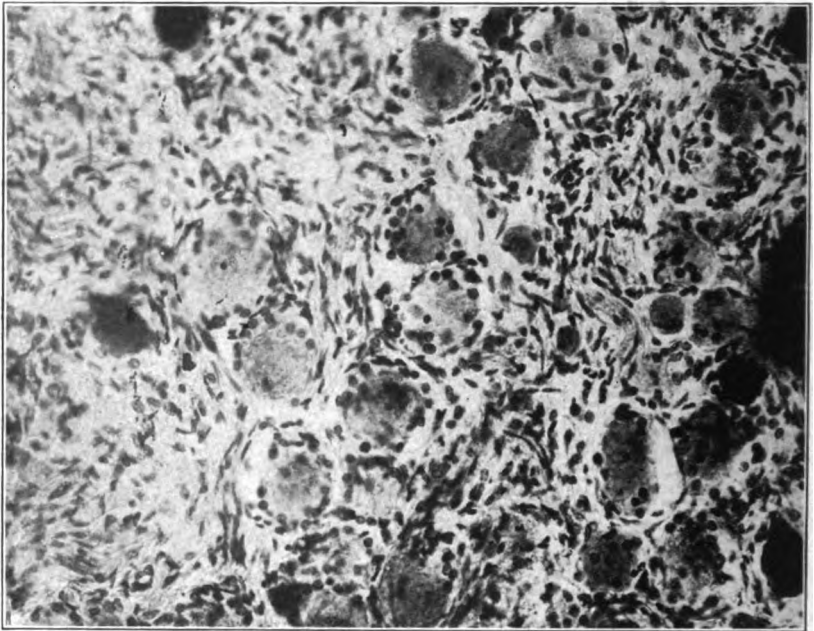


Fig. 7.—Posterior spinal root ganglion, high power. Case 2: showing faint diffuse staining of ganglion cells, increase of interstitial cells and marked neuronophagy.

in a very sketchy fashion by Major Young, Canadian Army M. C., of No. 4 Canadian Casualty Clearing Station, who observed the case. Hence the histories will be given very briefly from incomplete notes. In both cases the spinal cords together with posterior nerve root ganglia were preserved in liquor formaldehydi. Part of the material was later treated with Zenker's fluid. The blocks were embedded in celloidin and the section stained with hematoxylin-eosin, cresyl violet, iron hematoxylin and by the von Gieson, Mallory's anilin blue and Unna-Pappenheim methods.

and packed with red blood cells (active hyperemia). Around these dilated vessels there was to be seen considerable free-lying blood, but it is uncertain that this was not a postmortem phenomenon occurring at the time the cord was removed.

Quite the most striking change was seen in the arachnoid. This membrane, usually quite thin and difficult to demonstrate, was markedly thickened. This thickening consisted of marked hyperemia of the small blood vessels, edema of considerable degree, hemorrhage into the tissue spaces, thickening of the connective tissue fibers, formation of new fibers and young connective tissue cells and an amorphous stuff that appeared to be cell detritus (Fig. 1). In none of the sections was there an infiltration of other blood elements, except the red blood cells. In no place were there to be seen the layers and

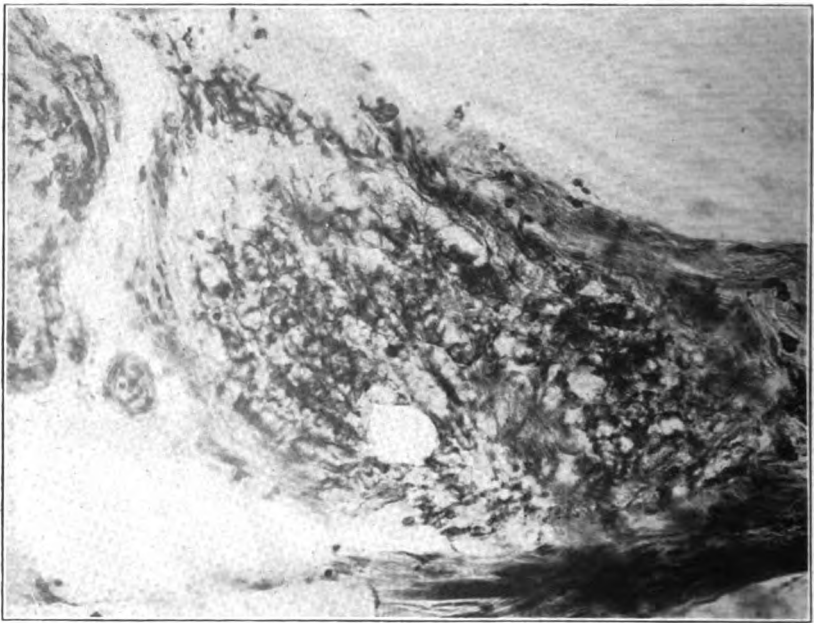


Fig. 9.—Cross section, anterior root just after emergence from the cord, high power. Case 2: showing complete degeneration of all the fibers.

clumps of polynuclear leukocytes as in septic meningitis, nor the lymphocytes and other related cells that form so prominent a part of the picture in tubercular meningitis, syphilis and poliomyelitis. This thickening of the arachnoid is always more marked over the anterior and anterolateral aspects of the cord than over the posterior. Often it is marked around the nerve roots that lie in the arachnoid and especially those around the anterior roots. In places the process has progressed so far that the arachnoid is so closely adherent to the pia that no line of separation can be determined and, more rarely, all three membranes appear to have fused into one, as though the inflammatory arachnoid had acted as cementing structure between the pia and dura.

The pia mater appeared rather thickened, mostly from swelling of the connective tissue fibers and partly by adhesion to it of the arachnoid. The

The Ectodermal Elements: The superficial layer of neuroglia lying just under the pia showed no changes. All neuroglia fiber, in so far as they were brought out with the stains, appeared normal in appearance and number. The cellular neuroglia showed considerable change. In the central gray, especially in the anterior horns, the neuroglia cells were greatly increased in number. These new cells had the round or oval nuclei, either darkly stained or showing the definite reticulum and the small, at times invisible, cytoplasm of young glia cells. These are the cells that Bashford calls "small round cells" and to me they appear as being entirely of neuroglia origin. In some places they are rather more numerous around some anterior horn cells (Fig. 3). Neuronophagy, as described by Bashford, was not seen. In the white matter of the cord the neuroglia cells of the anterior and posterior roots, as Bashford described, were increased.

In the posterior spinal root ganglions the interstitial cells of neuroglia origin were likewise markedly increased in number and tended to group around certain of the nerve cells. No neuronophagy was seen (Fig. 4).

The Nerve Cells: The anterior horn cells showed but little change in this case. In most of the cells the tigroid substance appeared to be more finely divided than in normal cells, even for cells fixed in liquor formaldehydi. In some cells the nucleus was rather eccentrically placed, and the tigroid was lessened in amount, especially in the center. It is difficult to say whether this was central chromatolysis due to destruction of the nerve fiber or the result of direct toxic action on the cell itself. While the picture may be looked on as pathologic, it surely must be in a very early stage (Fig. 5). As in Bashford's cases, so in mine, some of the cells of the endogenous cord fibers appeared altered in a similar manner as the motor cells, but always to a lesser degree.

The sensory cells of the posterior spinal root ganglions showed little if any change. In some section many of the cells appeared to take cresyl-violet rather faintly and the tigroid was very finely divided, but such pictures may be obtained at times with normal cells fixed in liquor formaldehydi. At any rate, there were no evidences of a real cell destruction. Some of the nuclei were eccentric but hardly more so than is often seen in normal material.

The Nerve Fibers: For some reason no pieces of the peripheral nerves were removed at necropsy. However, the nerve changes which were observed in the nerve root fibers were so striking and corresponded so closely with the nerve changes described by Bashford that enough can be seen in them to account for the symptoms. The blood vessels of the nerve roots were all widely dilated in marked hyperemia and in a number of cases hemorrhages had occurred. Degeneration of varying degrees was the ruling picture. In many places the nerve fibers showed signs of degeneration where they passed through the pia and just inside the cord. In sections stained by the Marchi method was seen very marked degeneration in the posterior nerve root fibers where they run in the arachnoid and more especially just after they have entered the spinal cord. These fibers lay as a black band in the root entrance zone of the posterior columns, by far the most striking thing in the section. In every case the maximum degeneration was to be seen in the fibers just after they leave the cord and begin to be enveloped by the arachnoid. The degeneration was so marked here that it would appear that this was the point of most extensive involvement of the peripheral nerves. The type of degeneration at this point appeared decidedly toxic while in the fibers, both above and below this point, the degeneration appeared to be of the

Wallerian, secondary type. Where the nerves enter the arachnoid, the degeneration was usually pretty complete. Some swollen axis cylinders were to be seen, but most of them had disappeared. The myelin sheath was completely gone. Many of the Schwann cells disappeared and the connective tissue trabeculae appeared to be diminished. Further peripherad the degeneration was of more moderate degree. Here also, on longitudinal section the axis cylinders were swollen, knobby and broken. The myelin had broken up into large and small droplets. The neurolema was shrunken and the Schwann cells were much increased in number (Fig. 6). These changes are regularly more marked in the anterior roots and always very much slighter in the posterior roots. This corresponds very well with the clinical side of the picture in which the motor signs are always more marked than the sensory. Outside the dura the posterior roots near the ganglions show little, if any, alteration from the normal.

Summary of Histologic Findings.—The histologic findings were:

1. Hyperemia, hemorrhage edema, and fibrous swelling in the arachnoid; thickening of the pia.
2. No changes in blood vessel walls; no round cell infiltration.
3. Increase of the cellular neuroglia in the central gray, around the root fibers and in the posterior root ganglions.
4. Evidence of beginning degeneration of both a secondary and primary character in the anterior horn cells and some tract cells. Hyperemia of the central gray.
5. Marked degeneration of primary and secondary character of the nerve fibers where they lie in the arachnoid, always most marked in the motor fibers.

CASE 2.—History.—The history of this case is very incomplete, having been given to me from memory by Major Young, C. A. M. C., at a time when all the hospitals in Flanders were extremely busy with the wounded from the Passchendaele battle. The patient was admitted to No. 4 Canadian C. C. S. about the middle of August, 1917, with a flaccid paralysis of both legs. The next day both arms were similarly paralyzed and later both seventh nerves were involved. The patient died about a week after the onset of the paralysis. A necropsy was performed by Captain Stokes, R. A. M. C., pathologist to No. 10 C. C. S. at Remy Siding, who kindly gave me a very generous share of the spinal cord segments and posterior root ganglions.

Histologic Findings.—*Mesodermal Elements:* The dura mater appeared to be perfectly normal in the few pieces at my disposal.

As in Case 1, the arachnoid showed marked changes but they were less easy to demonstrate on account of poor preservation due to the fact that the cord was not removed with the dura intact. However, one can see, in places, the hyperemia, edema, hemorrhage, swelling of the connective tissue fibers and cell detritus described in the previous case.

The pia mater is edematous, hyperemic and thickened somewhat more so than in Case 1. There is no infiltration with lymphocytes or leukocytes anywhere.

The blood vessels show a hyperemia in the gray matter of the cord but to a much less extent than in Case 1. The vessel walls show no change, and there is no infiltration of the Virchow-Robin spaces with lymph or other cells.

The Ectodermal Elements.—The fibrous neuroglia appeared normal, but the cellular neuroglia was increased in the central gray matter and around

the nerve roots to the same extent as in Case 1. There was no true neuronophagy. In the posterior root ganglions the neuroglia cells were markedly increased in number and neuronophagy was seen in many places.

The Nerve Cells: Many of the anterior horn cells appear quite normal while others show fine granulations of the tigroid and probably beginning cloudy swelling. The tract cells, as the motor, show on the whole less change than do those of Case 1.

The sensory cells of the posterior ganglions show, however, much more marked changes. The cells appear swollen and stain very pale. The edges of many are indented and phagocytic glia cells are to be seen invading the cytoplasm (true neuronophagy). Some of the cells stain diffusely dark and also show neuronophagy (Fig. 7).

The peripheral nerve fibers of the roots show even more degeneration than in Case 1, always more marked in the anterior roots. Just after these fibers emerge through the pia they often show a very extreme degeneration of a primary type (Figs. 8 and 9). Further peripherad in the arachnoid extreme degeneration is seen in longitudinal section—broken, swollen, knobby axis cylinders, disappearance of myelin, increase of Schwann cells and shrunken neurolemma. The extra dural portion of the posterior roots near the ganglions show a very interesting picture of moderately advanced degeneration. The axis cylinders appear rather normal. The myelin substance has broken up into large and small droplets still lying in the Schwann cells. The cytoplasm of the Schwann cells has apparently increased somewhat in amount and takes the stain darkly, showing a definite network between the fat droplets, giving a mosaic like appearance to the whole. This is the so-called "neuro-keratin network" described in normal and beginning degenerating nerves (Fig. 10).

Summary of Histologic Findings.—The histologic findings were:

1. Hyperemia, hemorrhage, edema, swelling in the arachnoid; thickening of the pia.
2. No changes in the blood vessel walls; no round cell infiltration.
3. Increase of the cellular neuroglia in the central gray, around the nerve roots and in the posterior root ganglions.
4. Slight secondary and primary degeneration in the anterior horn and a few tract cells; slight hyperemia of the central gray.
5. Marked primary and secondary degenerations in the anterior nerve root fibers, just as they emerge from the pia and where they run in the arachnoid.
6. Beginning degeneration in the posterior nerve roots just outside the dura.
7. Marked degeneration of the posterior root ganglion cells with neuronophagy.

PATHOGENESIS

It is quite obvious that the cases reported here and in previous articles represent a true disease of the nervous system which, until the publication of the article of Bradford, Bashford and Wilson, had never been accurately described. The way it affects the nervous system appears to be quite different from that of other acute diseases. The process is diffuse, although the peripheral system is always more severely damaged than the central. In the vertebral canal, not only the spinal cord, but also its membranes participate in the process. It is doubtful how the organism and its products reach the nervous system

where the changes occur, and from which the organisms have been recovered. The fact that the nerves are more severely affected at the arachnoid level than more peripherally would seem to indicate that the path of infection is not along the nerves.

The portal of entry must be by the blood or lymph channels. It is difficult to choose between these two, principally because of the fact that there is no lymphocytic proliferation. What evidence there is points rather to the lymphatic conveyance. Bashford states that in his monkey cases "the only constant features—outside of the nervous system—were considerable hyperplasia of all the lymphatic glands, and some congestion at the bases of the papillae of the kidneys." Wilson succeeded also in recovering the organism from the cervical lymphatic glands in the monkey. However, Key and Retzius have shown that pigment injected into the subarachnoid space is to be found later in the cervical glands; hence, one must consider the possibility that these organisms may just as well have gone from the central nervous axis to the cervical glands as the other way. Certainly this is the direction of drainage.

However, when the organisms reach the central nervous system they produce there two definite changes: 1. In the mesodermal elements of the arachnoid and pia the result is edema and hemorrhage, with some increase of connective tissue cells, without any of the usual signs of inflammation, i. e., infiltration with white blood cells and lymphatic elements. The lymph spaces of the spinal blood vessels show no changes. 2. The changes in the ectodermal elements appear to be most marked in the neighborhood where the greatest mesodermal change takes place, i. e., in the arachnoid where the nerve roots run through it. These nerve fibers here show marked degeneration of a toxic and infectious nature. In the peripheral nerves the degeneration may be looked on as both secondary and toxic in nature. In the spinal cord and posterior root ganglions we see the usual picture of infectious-toxic degeneration, with some evidence of early secondary degeneration due to the destruction of the fiber in the arachnoid. The increase of the neuroglia cells, the degeneration of the nerve cells and the neuronophagy are all parts of the infectious-toxic picture. That the changes observed in Case 2 were, on the whole, more severe than in Case 1 is explained by the fact that the disease had had a longer course. In Bashford's cases where the changes were more severe than mine, this is probably due to the same cause.

The question of the proper name for the disease is still an open one. Gordon Holmes called it "acute infectious polyneuritis." Bradford, Bashford and Wilson have employed the same term with considerable

misgivings in the light of the central nervous system changes. Kennedy, recognizing the inaccuracy of the term, has suggested the name "acute neuronitis," not without misgivings on his part. The present writer is at a loss to name the disease properly. To call it an acute arachnoiditis appears very inadequate, and hence he has been obliged to fall back on the clinical manifestations of the disease for the title of this paper. Perhaps the term "acute infective meningomyeloneuritis" might be acceptable.

INFECTIVE NEURONITIS *

FOSTER KENNEDY, M.D., F.R.S. (EDIN.)

NEW YORK

During the spring and summer of 1917, and to a lesser degree in 1918, I was privileged by circumstances in France to see a considerable number of cases having many of the easily recognizable symptoms of acute polyneuritis, that presented unmistakable evidences of involvement of the spinal roots and of the central nervous system as well.

Detailed reports of some of these, together with the necropsy material of two fatal cases that had been clinically carefully examined, I gave to Sir John Rose Bradford who collaborated with Captain Bashford and Captain Wilson in examining them along with other material arriving subsequently from other areas.

The brilliant results of these joint investigations have recently arrived in this country¹ and make it unnecessary to put before you an extended succession of clinical descriptions of this disease.

Sufficient stress, however, has not been laid on the clinical manifestations in these cases of the widespread affection of nervous tissue other than the lower motor neuron.

I propose, therefore, to describe certain cases under my care revealing definite clinical variants from the syndrome of polyneuritis as we have known it, which, when considered along with the widespread changes in the posterior ganglions, spinal roots, ventral cornual cells and Betz cells of the cortex, would seem to make it fitting to remove this disease from the neuritides proper, and to designate it by a title more descriptive of the clinical and pathologic picture produced.

REPORT OF CASES

CASE 1.—Private, J. J. W., Twenty-Fifth Northumberland Fusileers, when in good health suffered prolonged burial on May 28, 1917. He was shaken by this experience; when being sent down the line he had severe headache, and was tremulous and nervous; he complained of pains in his back and limbs and for four days had a mild rise of temperature. He then recovered from these generic symptoms, was made a temporary orderly in hospital, and did his work well and enjoyed it.

On July 14 he felt well and worked as usual. When handing out dinners in a ward he suddenly felt weak "in the thighs, not in the feet." He lay

* Read before the Meeting of the American Neurological Association, at Atlantic City, N. J., June 16-18, 1919.

1. *Quart. J. Med.* 12:88, 104 (Oct.) 1918 (Jan.) 1919.

down for two hours, after which he found himself hardly able to stand and experienced numbness and tingling in the toes; he found that he "could not feel his blanket with his toe ends."

At this time also he became conscious of similar sensations in the fingers. In a few hours he developed acute headache and shooting pain in the back of the neck, and aching pains in both knees. During these hours the feeling of numbness gradually crept up to the knees.

Next morning he had great difficulty in standing and found his finger tips anesthetic when washing. On that day the knee jerks were very brisk and the ankle jerks were absent. There was great weakness of the movements of both thighs and to a less relative degree of dorsiflexion of the feet. The arms, especially in shoulder movements, were weak; the face and palate were normal. He could, with difficulty, walk alone. Pinprick and touch could not be felt below the root of the toes on both feet and the middle of the first phalanx in the hands.

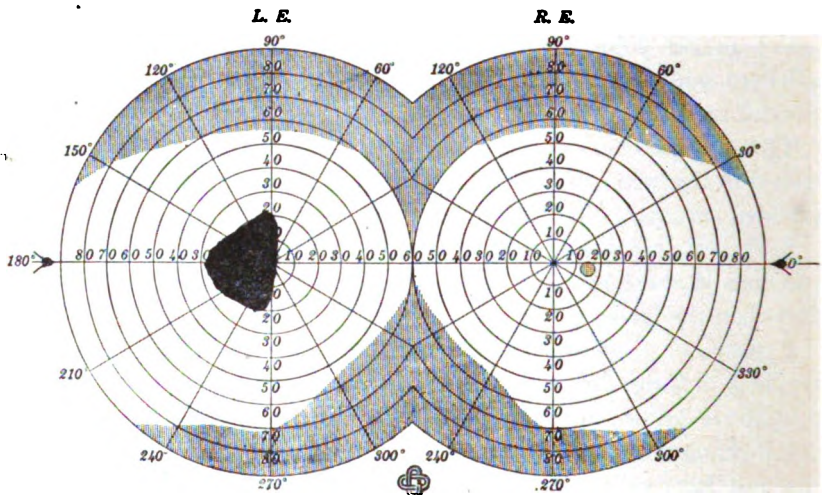


Fig. 1 (Case 1).—Left paracentral scotoma.

On the following morning the knee jerks were absent, the palate and diaphragm normal, and there was visible a very slight weakness in closing the left eyelid and in retracting the left angle of the mouth. The upper limit of analgesia was now across the dorsa of the feet and the middle of the hands, and there was progressive loss of strength in the hip and shoulder movements, without any corresponding change in distal power. On the afternoon of the same day he could not close the left eye, and he complained of inability to taste his food and of numbness in the mouth and lips. He was seen to have lost appreciation of pinprick and touch in both fifth nerve areas. The calf muscles were then very sensitive to pressure.

The next day (July 17) brought to him great difficulty in swallowing solids but not fluids—which difficulty was referred by him to below the level of the thyroid cartilage. (All these patients volunteered a description of this symptom, complaining of "a lump" being felt in the upper esophagus.) Dorsiflexion of the feet was still good, but gluteal, psoas and adductor actions were practically abolished. The right thigh was less affected than the left. The

muscles lacked tone, but were not wasted. He could feed himself easily because the hands were still strong, but movements of biceps, triceps, deltoid and trapezius were almost quite ineffective on both sides.

The zone of sensory loss extended below the upper third on the legs and forearms. He complained of difficulty in passing water, of not feeling the act of micturition and of not feeling the bedpan properly. Examination thereupon revealed an almost complete loss of sensation over a saddle-shaped area on the buttocks, and over the penis and scrotum as well. The diaphragm was hardly in action; the abdominal rectal muscles were very weak; he was quite unable to turn over in bed, and no abdominal reflexes could be obtained.

Five hours after this examination—on July 17 at 11 p. m.—the border of loss to pinprick and touch was in the middle of the thighs and the lower third of the upper arms. Deep muscle pain sense in the calves, sense of position in the toes, ankles, fingers and wrists, together with vibration sense in both tibiae, were all absent. The palate remained unaffected, while swallowing became progressively more difficult. Coughing became troublesome; there was also progressive weakness in getting rid of phlegm properly. He was unable to move at the hips or knees; the ankle and toe movements were present though feeble.

On the following morning, seven hours later, he was astonishingly better. He swallowed more easily. The diaphragm acted well. He could feed himself again, and could draw the legs up with fair power. Urine was passed with less delay than before, and he was dimly aware of its passage through the urethra. The sensory loss in the sacral root areas was still present but was less deep. The fifth nerve change was stationary, but the stocking loss of sensation was quite definitely reduced to below a sharply demarcated line two inches above the malleoli. On this day of improved condition the visual fields were mapped out, and a paracentral scotoma was found on the left side, accompanied by disk pallor and subjective loss of visual acuity in the left eye. This extraordinary rally was maintained for just ten hours when he suddenly began to feel weak, and in fifteen minutes lost all power of movement in the legs; inside of an hour he lost the power of phonation, and began to have intense difficulty in swallowing. There was great discomfort referred to below thyroid cartilage. The pulse rate was 134. For the first time there was weakness of the right side of the face. His voice could only be raised to a faint whisper, but he asked for a pencil, which was used clumsily but legibly. The diaphragm was not paralyzed; the palatal reflex was brisk; recti abdominis, fair. He could move his toes and ankles fairly well, but there was no visible movement achieved at the great joints. There was urinary retention and sacral sensory loss as before, but only the toes were then anesthetic, and there was no loss to pinpricks on the hands.

On July 20—the next day—the right side of the face was entirely paralyzed; otherwise his condition was somewhat better. He could occasionally phonate. The vocal cords were in cadaveric position and almost quite fixed. The corneae were anesthetic, the pulse slower and intermittent. Consciousness was acutely clear.

On July 21 he became cyanosed and died of asphyxiation. Blood cultures—by the ordinary methods—were in this and two other cases negative.

Examination of the cerebrospinal fluid was negative on the third day of his acute illness, at which time he had a leukocytosis of 14,000. Sections of the cortex, medulla and cord, cervical and lumbar enlargements with root ganglions and certain peripheral nerves were sent to Captain Bashford, to whose findings reference will be made later.

As outstanding features of this case one must notice: first, generalized peripheral neuritis; second, spinal root involvement as shown by diminished sphincter control and loss of sensation in the areas of distribution of the lower sacral segments; third, the changes in the posterior tracts which abolished appreciation of pressure on muscles, previously very sensitive in this regard, and which caused the senses of vibration and joint position to disappear.

The greater incidence of the disease on the proximal rather than the distal muscles is quite unlike the usual clinical picture of polyneuritis and is, I believe, due to the imposition of a palsy of ventral root and ventral horn origin on one proceeding from a patchy degeneration of peripheral nerves.

The constant paralysis of the face, and the constant sensation of obstruction in the throat may be regarded as due to specific action of the infecting agent and so are analogous to trismus in tetanic, and oculomotor palsy in diphtheritic infections.

CASE 2.—Private H., Fifteenth Canadians, gave a history of having had "P. U. O." or "Trench Fever" some weeks before the onset of numbness and tingling in the hands and feet. When seen on Dec. 16, 1917, the right side of his face was paralyzed, his voice was very weak and the vocal cords were almost immobile in the cadaveric position. He experienced great difficulty in swallowing although the palate and its reflex were normal. There were extensive flaccid palsies around the shoulder joints, more grossly evident on the right than on the left side, and there was weakness but no paralysis of the hands. He was unable to turn in bed. The upper recti abdominis were definitely stronger than the lower recti. There was complete flaccid palsy at both hip joints, and almost complete palsy of both feet. He was quite unable to tell when micturition had begun or had ended. There was frequent bladder incontinence. All other spinal reflexes were lost. There was total loss of sensation in the fifth, sixth and seventh cervical root skin areas, and much relative loss in all the sacral root skin areas. My notes contain no mention, nor does my memory tell me of sensory defects referable to peripheral nerve change such as were described in the first case.

For the privilege of having seen this man and also the following I am indebted to the courtesy of Major Clayton, R. A. M. C.

CASE 3.—Second Lieutenant C. E. C., Thirty-Third Battalion, Australian Imperial Force, was utterly exhausted at Paschendaele in October, 1917; he was ill and miserable, but did not go off duty. A month later he had what was thought to be influenza; three days afterward weakness in the legs developed, and within a few hours he was quite unable to stand. When I examined him a month later I found complete facial diplegia, difficult deglutition and as usual, a normal palate. There was flaccid palsy round the great joints of all four limbs with a high degree of atrophy in the gluteal and thigh muscles; all movements of the toes and ankles were retained and were of fair power. The trunk muscles were very weak, but the recti abdominis were strong and the abdominal reflexes were retained while all others were absent.

Pinprick and touch were not appreciated in the second and third lumbar root areas, but were acutely felt in the legs and over the sacral region. In over a month's illness he had only once lost control of his bladder.

CASE 4.—The last case which it seems necessary to describe is that of Corporal H. H. E., Royal Army Service Corps, Divisional Train, who gave the usual history of a febrile attack in March, 1918, through which he had

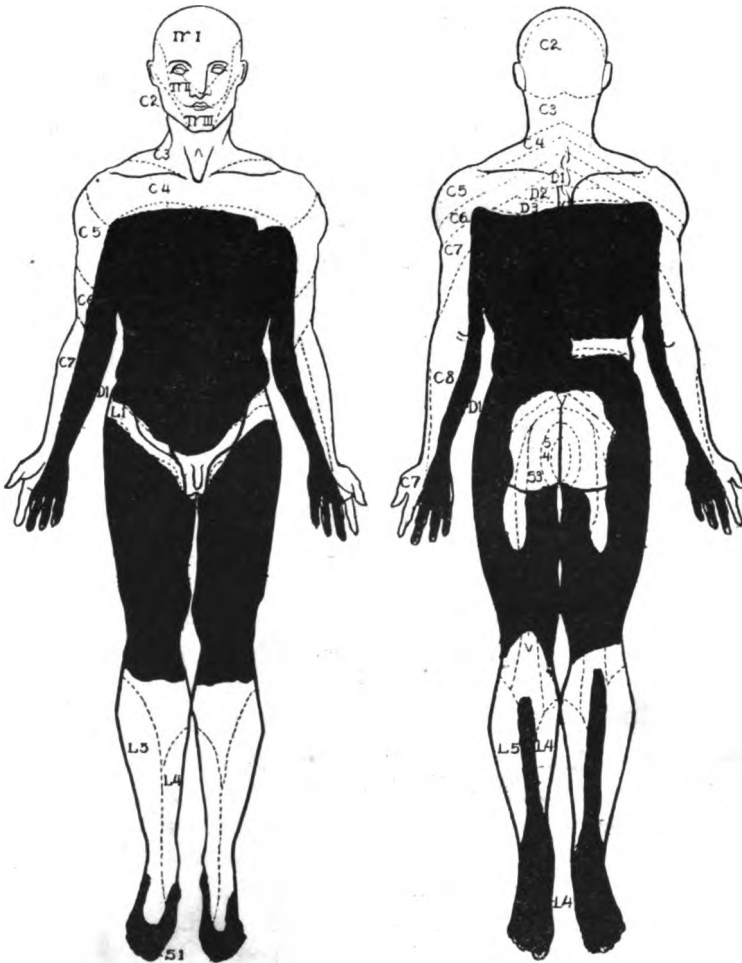


Fig. 2 (Case 4).—Shaded areas represent sensory loss.

remained on duty. Five weeks later numbness and tingling in the feet and legs and hands supervened. These were slowly progressive so that when I first examined him he was quite unable to walk. At the same time he showed an incomplete paralysis of the left face, and complained frequently of a feeling as though he had a lump in the throat although swallowing and speech were both functionally intact. The shoulder muscles were reduced in power, the grasps very weak. The trunk muscles were much enfeebled so that he could

not turn in bed without help. The thigh muscles were weak and flaccid and the glutei weaker and still more flaccid. The muscles below the knee were reduced in power to a much smaller extent.

The abdominal reflexes were lost. The knee jerks faintly present, the ankle jerks brisk and the plantar reflexes were both of extensor type. The sphincters were never affected, but no erections occurred, and all sexual desire was absent during his illness.

The sensory changes were remarkable and varied little in amplitude and not at all in distribution during the two and a half months he remained in hospital in France. There was almost complete insensitiveness to pinprick, touch and temperatures in the seventh and eighth cervical root areas, in all those served by the dorsal roots, and those supplied by the second and third lumbar and the first sacral roots; that is to say, from the seventh cervical segment downward, all the posterior root areas were almost quite defunctionated with the exception of the first lumbar, the fourth and fifth lumbar, and the second, third, fourth and fifth sacral areas, which were intact so far as repeated examinations revealed.

There was no loss of sense of position; deep muscle pain sense was certainly nowhere increased and was probably below normal in affected regions. The vibrations of a tuning-fork were not felt in the toes but were felt faintly on the patellae. They were, however, clearly felt on the tibiae and iliac crests. In affected areas, appreciation of compasses was absolutely bad, and elsewhere normal. The cerebro-spinal fluid was unchanged and the Wassermann test was negative.

While in my earlier cases the main incidences of the disease fell peripherally, with occasional signs of root involvement, produced probably by ascending lymphogenous extension, the last case emphasized the necessity of including something more than mere peripheral change in our conception of the condition as a morbid entity. Though its syndrome was necessarily determined somewhat differently from that of the others, yet its identity of origin, as shown by the facial, pharyngeal, humeral and other palsies, is indubitable; it displays nudely a clinical picture which was in the others, but which was overlaid as in a palimpsest by the detail of a generalized polyneuritis.

A study of the sections made by Captain Bashford revealed a patchy neuritis in the peripheral nerves, and a degeneration of the cells in the ventral and dorsal cornua, and especially in the cells of the posterior ganglions. Similar, but more benign changes, were found in the deeper layers of the cerebral cortex and in the cells of the pontine nuclei. A small round-celled infiltration was found around the ganglion and cornual cells, but never around the meningeal vessels and capillaries as is usual in poliomyelitis; this was, in Bashford's opinion, a much later phenomenon than were the degenerative changes in the nerve cells themselves.

The ependyma of the spinal central canal showed constant extensive proliferation. The meninges were normal.

Small quantities of an emulsion of affected spinal cord preserved in glycerin injected into monkeys subdurally produced the disease clinically after an incubation period of from five to seven weeks, and histologic examination of the nervous tissues of these animals revealed conditions essentially identical with those described in man. The disease further was reproduced by inoculation from monkey to monkey.

Nervous tissue from fatal cases and also from monkeys affected with the disease as has just been described were investigated bacteriologically by Capt. J. A. Wilson, R. A. M. C., who succeeded in obtaining positive cultural results by following the methods adopted by Flexner and Noguchi in their search for the organism of poliomyelitis. It suffices to state here that, under strict anaerobic conditions, yellowish colonies appeared capable of subculture composed of minute rounded bodies arranged irregularly or in pairs.

This organism inoculated subdurally into a monkey reproduced the disease clinically and pathologically, and was recovered later from the nervous tissues of the animal so inoculated.

In conclusion, stress must be laid on Wilson's observation of the ease with which the organism was recovered from the cerebral cortex—a consideration that lends emphasis to the present contention that here we have to do with a widespread neuronc infection and not with a condition limited to peripheral twigs.

HEREDITARY OCCURRENCE OF HYPOTHYROIDISM WITH DYSTROPHIES OF THE NAILS AND HAIR *

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The study of individuals and families that may show various manifestations of mental and nervous degeneracy not infrequently discloses the existence of constitutional disorders that are direct or indirect causes of these.

These constitutional disorders appear sometimes as peculiar defects or abnormalities of body structures that tend to be transmitted through the family. Numerous instances of these are recorded in the literature dealing with problems of heredity. These are in most instances treated rather more from the point of view of being specific unit qualities that are transmitted than as the results of a more general inherent deficiency of organs whose healthy functioning is essential for determining normal development and successful adaptation to the requirements of life.

Recently we have had the opportunity of studying a family that for generations had shown among its members peculiar trophic disorders of the nails and hair, and various abnormalities of a mental or nervous character.

While the more obvious abnormalities of this group are of special dermatologic interest, the fact that these seem to be fundamentally related to some constitutional disorder of an endocrinopathic type that peculiarly affects the nervous and mental make-up of the members of this family, makes the entire subject one of much importance for neuropsychiatry.

Our acquaintance with this family began with a boy who, at the age of 18, had been brought into the Juvenile Court of Detroit because of several instances of petty thieving. The frequency of his delinquencies and certain mental traits suggested the existence of feeble-mindedness and led to his admission for observation to the Psychopathic Hospital at Ann Arbor.

REPORT OF A CASE

Examination.—The physical appearance of the boy was quite abnormal (Figs. 1 and 2). He was about 5 feet, 5 inches tall, and weighed 138 pounds. He appeared unusually well nourished. The skin of the face, trunk and

*Read before the meeting of the American Neurological Association held at Atlantic City, N. J., June 17-18, 1919.

extremities was plump and felt as if it were thickened in its deeper parts. Around the eyes the skin was puffy as if from increased subcutaneous fat. The hands were chubby and the fingers were short and clubbed. The hands and feet were cyanotic. The surface of the skin lacked moisture. The skin of the finger tips and soles of the feet was exfoliated in places.

A very striking feature in his appearance was the scant amount of hair on the head and body and peculiar abnormalities of the nails. While the head seemed fairly well covered by hair, this on close inspection was seen to be unusually thin, and there were patches in which the scalp was covered only by a fine lanugo. The eyebrows were reduced to a few scattered hairs and these were stiff and short. There was but little hair in the axillary and pubic regions and the latter conformed to the feminine type of arrangement.

The nails of the fingers and toes all lacked a quarter or a half inch of reaching the tips of the fingers or toes. There was no lunula. Their free margin was thickened and broken. Their surfaces were smooth and not ridged. The nail bed was exposed and in places showed suppuration (Fig. 3).

The pulse rate was 80; the blood pressure 130. There were no cardiac abnormalities. The body temperature was always a little below normal. On several occasions it was 96.8 F.; usually it was under 98. The Wassermann reaction test was negative on the blood. The urine at admission had a specific gravity of 1.010 and showed a few hyaline and granular casts. In other respects it was negative. The roëntgen-ray examination showed a normal sella turcica.

There were a number of neurological abnormalities. There was a frequent tic-like movement of the face, and when he became emotionally excited there was a tic of the right shoulder. His speech was somewhat thick and nasal in quality. Bone conduction for sound was absent in the left ear. The left pupil was larger than the right. The fundi of the eyes were normal. Refraction tests showed a high degree of hyperopia. The form field of the left eye showed a retraction of the field for white. There were no paralyses or disturbances of sensibility. The fingers showed a fine tremor on extension.

His mental reactions were slow and he showed a definite intellectual defect. Psychometric tests gave him a mental age of 12 years and 6 months. His attitude toward his delinquencies was immature and unreasonable. In other respects he showed few definite peculiarities.

The general appearance of the boy, the peculiar condition of the skin and the dystrophies of the hair and nails suggested the existence of a myxedematous condition. This view was confirmed by a biopsy of the skin kindly made by Professor Wile of the department of dermatology.

The patient was tested for glucose tolerance and it was found that he could take 250 gm. of glucose in twenty-four hours before sugar appeared in the urine.

Treatment.—He was put on thyroid extract in 5 grain capsules three times a day. At the end of twenty-seven days there was increased perspiration. The exfoliation of the skin of the hands and feet ceased. The hair was less fragile and there was a very noticeable increase in the hair of the scalp and eyebrows. The nails appeared to be improved and they seemed to show some growth. Subjectively he appreciated an improvement in his general feelings. After a month of thyroid treatment he began to show toxic effects; the pulse rate was 110. Treatment was discontinued. The improvement of the skin condition at once ceased. The dryness and exfoliation again returned. On resuming treatment, improvement again occurred.

The clinical symptoms and their response to treatment in this case leave little doubt but that the disorder of the nails and hair were but parts of a constitutional disturbance due to hypothyroidism.

Family History.—The case became of increased interest through the fact that these same deficiencies had been present among members of the boy's family for at least six generations, and that in addition to the dystrophies of the hair and nails, both those affected in this way and many others without these defects showed various disorders of nervous functions (Fig. 4).

It has been possible personally to substantiate the accuracy of the data in this case in almost every instance. Our information regarding the family goes back to the great-grandmother of our patient, I—1 M. J. C. 1st. This woman lived in Canada and was of French extraction. It is known definitely to members of the present generations that she had the same characteristic abnormalities of nails and hair. A photograph in the family shows a marked facial similarity to her daughter, II—1 M. J. C. 2nd. It is probable that these abnormalities were present in still earlier generations. This is suggested in the reply that M. J. C. 1st gave to her daughter that these were the signs of an unhealthy generation. Information that we regard as trustworthy, but that has not yet been personally confirmed, is that a brother of II-1 has the abnormality and of his six children, three have the disorder, and one is insane.

II-1 married a man who was free from defect. From this marriage there were ten children. The oldest of these, a male III-2 had the characteristic defect. He married a woman without the defect, and from this marriage there were six children. The oldest, a female, L, IV-1, has normal nails and fairly abundant hair of the scalp, but few in her eyebrows. She is of short stature and has peculiar short, chubby fingers. She is of neurotic constitution, and from early childhood has had a severe facial tic. She has had no children. The second child, a female, A, IV-2, does not have the defect. She is either hysterical or epileptic. When a small child she would have attacks in which she was unclear. In these she would take hold of her mother and remark that the floor was sinking under her. Later on these ceased, but at the age of 16 she had attacks in which she was unconscious for a brief period. There were no convulsive movements. These have recurred from time to time during her adult life. It is of interest to note that although she has always had an abundance of hair on her head, in the spring of 1919 she had a miscarriage, and following this her hair fell out in large amounts. She is married to a man without the defect and has had two children. The first of these died in infancy from pneumonia, and the second during the first years of her life suffered from marasmus. P, IV-4, a male, was our patient. His physical and mental disorders have been noted previously. It is said by his family that he and his father closely resembled each other, not alone in the defects of nails and hair but in having a thick skin and in their general physical make-up. Since his discharge from our observation, this boy has been delinquent frequently, having been implicated in several instances of stealing, and at the present time his whereabouts are unknown.

Between the third and fourth child there were two miscarriages. The fourth child, a boy, G., IV-5, is now aged 19. He has thin eyebrows and a fair amount of head hair. His nails are normal. During an attack of typhoid fever he had a severe delirium and since then he has never been physically strong.

The sixth child, a boy, E., IV-6, has the characteristic defect of nails and hair (Figs. 1 and 2). He was first seen at the age of 7. His appearance

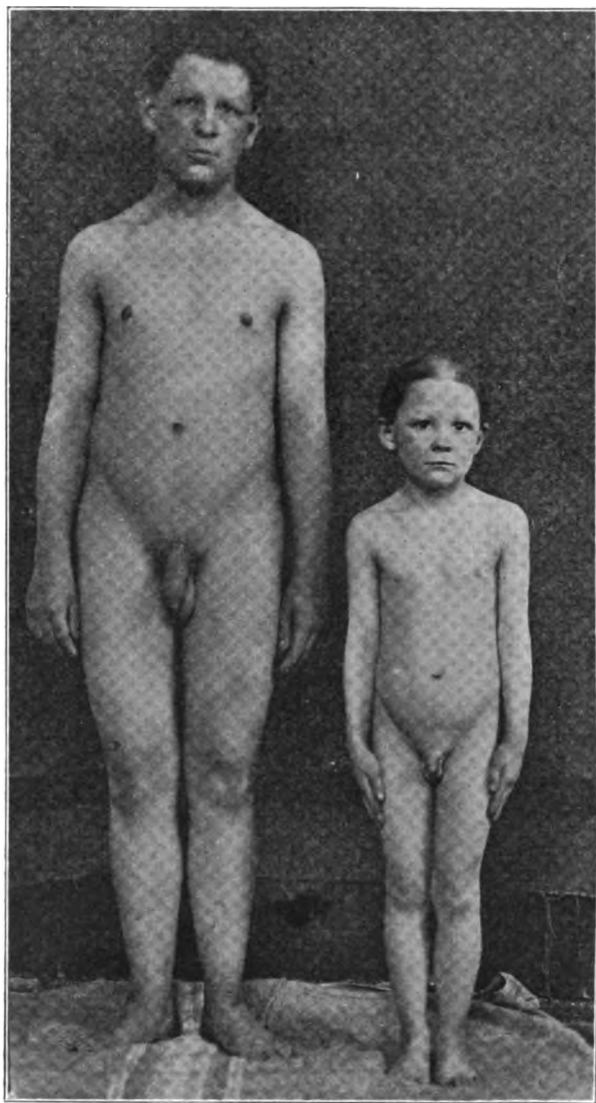


Fig. 1.—Patients IV-4 and IV-6.

at that time had a cretoid character. He looked like a little old man. His skin seemed myxedematous. There were patches of baldness on the top and back of the head and there were no eyebrows. Four years later his hair was uniformly scant over his head. There was a lateral scoliosis in the dorsal region. The thyroid gland was not palpable. There was a tic-like movement of the head and choreiform movements of the extremities. At the age of 13 he came into the courts because of malicious conduct and incorrigibility. He was sent to the State Training School for the Feeble-minded. Psychometric examinations gave him a mental age of 9 years and 1 month. He responded very little to thyroid treatment.

The seventh child, R., IV-7, does not have the defect, but is mentally subnormal.

The second child of the third generation, W., III-3, a male, is said to have had hair at birth, but none after early childhood. At the present time, he is completely bald. He has no eyebrows or eyelashes and no body hair. His nails are defective in the characteristic way. Between the ages of 25 and 32 he had typical epileptic convulsions. At the present time he has peculiar feelings as if an attack were coming on, but no further development. He married a woman free from the defect and has had four children. The eldest, a girl, M., IV-8, has the defect. Her head hair is limited to a thin downy covering. Her nails are defective and she has thick skin and clubbed fingers. Mentally she is backward, and is now in a special room. Her thyroid is hard and abnormally small. Menstruation first appeared at the age of 16.

T., IV-9, a boy, aged 12, lacks the defect in the characteristic type, but his hair is unusually thin. He has nocturnal enuresis. Mentally he is feeble-minded.

H., IV-10, a boy, aged 10, does not have the defect, but is feeble-minded.

J., IV-11, a boy, aged 5, has thin hair, but normal nails. He has nocturnal enuresis.

The third child of the third generation, J., III-5, lacks the defect. He has had a bad criminal record in the courts. He has twice married, but has had no children.

The fourth child, E., III-7, seems to have been quite normal as to hair and nails. She married a normal man and all of her descendants, for two generations, have been free from the defect.

The fifth and sixth children of the third generation were twins and lacked the defect. One of these, III-9, died in infancy. The other, E., III-10, married a normal man and had two normal children. She, herself, died from acute tuberculosis.

The seventh child, L., III-2, had normal hair and nails. She married, and has had eleven children. All of these have remained free from the defect.

The eighth child, J., III-14, has no hair on her head and her nails are defective. She married a normal man, III-15, who is also a brother of III-4. She has had seven children. The first of these, T., IV-30, was free from the defect, but died in childhood from scarlet fever. The second and third children lacked the defect, but died in childhood from marasmus. The fourth child, W., IV-33, died in infancy from meningitis.

The fifth child, M., IV-34, a girl, aged 13, has normal nails and hair, but her teeth are notched along the cutting edge. Mentally she is feeble-minded.

The sixth child, C., IV-35, has the typical defect. He has nocturnal enuresis and is feeble-minded.

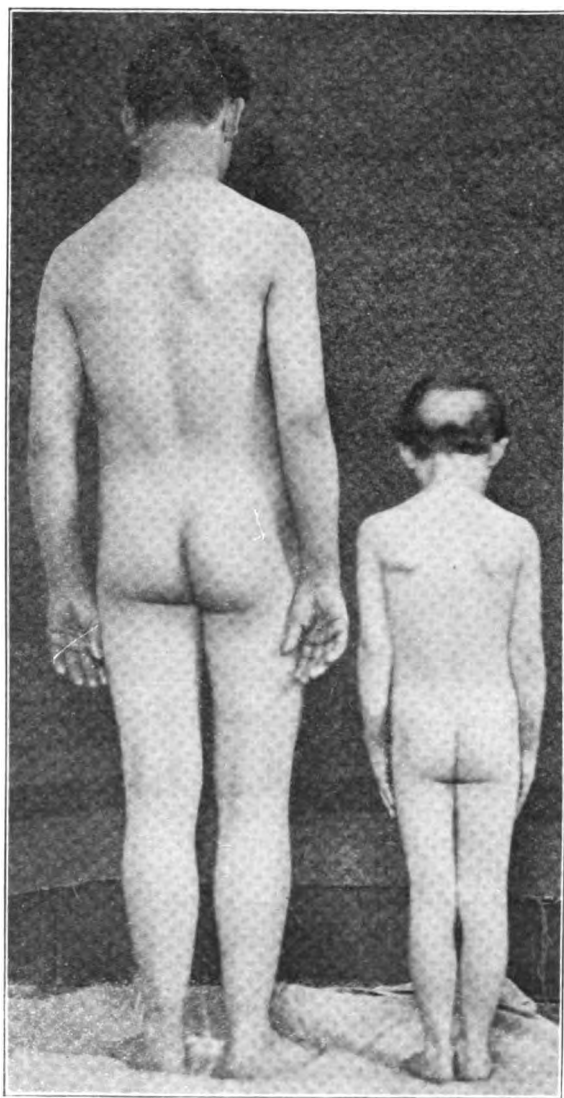


Fig. 2.—Patients IV-4 and IV-6.

The seventh child, M., IV-36, aged 7, has the family abnormality of nails and hair. She has nocturnal enuresis and is feeble-minded.

The ninth child of the third generation, M., III-16, lacks the defect. She married a normal man and has had nine children, all of whom are free from the defect and are of average mentality.

The tenth child, L., III-18, has the defect. Her teeth are worn away on the cutting edge. She married a normal man, and has had three children. The oldest of these, A., IV-46, has always had good hair, but his nails show the typical defect. He has some difficulty in hearing, but otherwise is healthy. The second child died in infancy and had abnormal nails. The third child, R., IV-48, has the defect. While the head seems fairly well covered by hair it is abnormally thin in amount. The teeth show irregularities of the cutting edge. Mentally he is subnormal.

Among sixty-one members of this family, belonging to six generations, the defect in its typical manifestations was present in fourteen instances. In these, both hair and nails were affected. The nail defect seemed to be about the same in all, but there was much variation in the

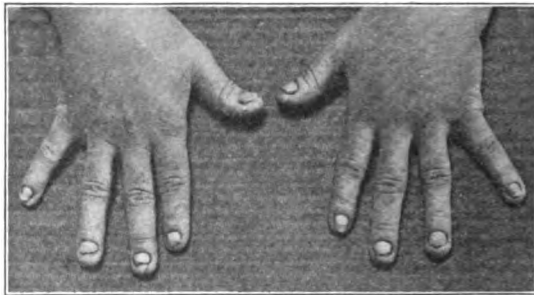


Fig. 3 (Patient IV-4).—Abnormalities of nails.

degrees of loss of head hair. In the greater number there was an extreme scantiness as to the amount of hair. In no instance was there a total loss of all hair of the head, the most extreme cases showing a fine lanugo-like covering of the scalp.

The defect tends to occur in a mendelian type of distribution, but the varied character of the abnormalities appearing in relation with the type defect of nails and hair is perhaps too complicated to be explained in a simple mendelian formula. The character of abnormal hair and defective nails behaves as a mendelian dominant. All persons that have the defect give a mixed progeny when crossed with normals. The defect never appears in a descendant who himself is free from the defect.

Aside from this particular abnormality, there are other features that show that in this family group there are conditions active in the production of a variety of disorders that are of much interest to neuro-psychiatry. This is the high frequency of feeble-mindedness and

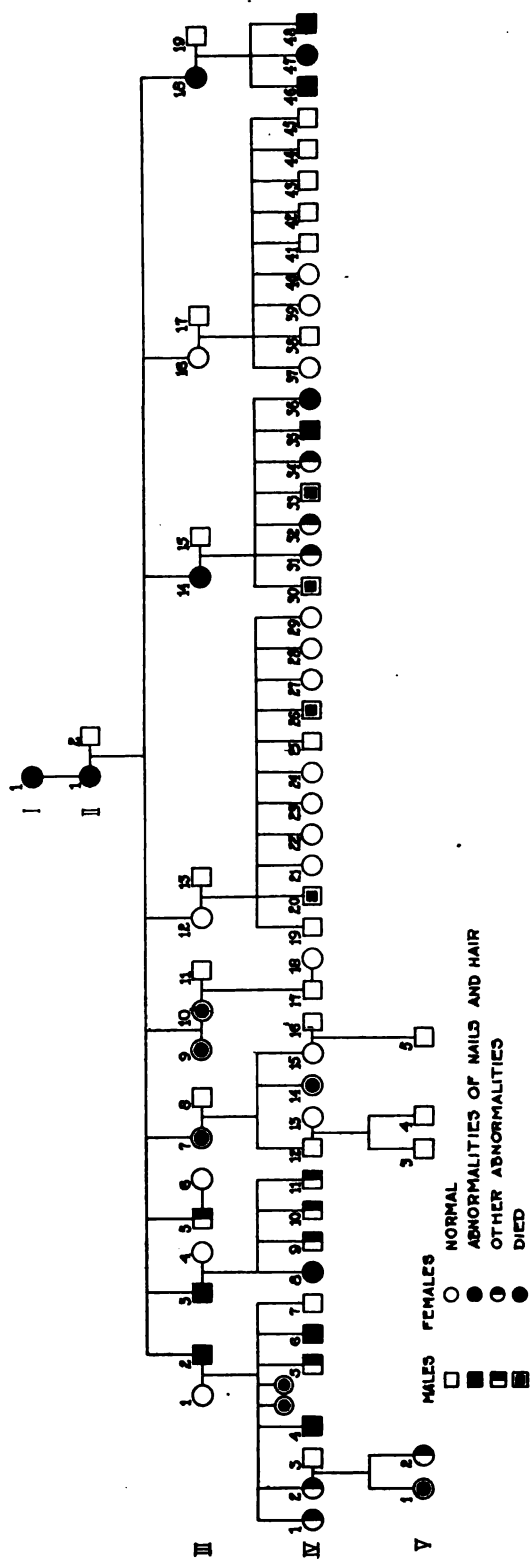


Fig. 4.

Heredity chart of family showing hypothyroidism and abnormalities of nails and hair.

neurological disorders of a degenerate type that are present among the family.

The members of the third generation who had dystrophies of nails and hair, and all of their descendants, numbered twenty-nine persons. Of these, twenty-two were definitely abnormal. Twelve of the latter had the characteristic family dystrophy, and ten others, who lacked this, showed other constitutional and nervous disorders. These included one case of epilepsy, one of hysteria, one of severe tic, four instances of feeble-mindedness, one of nocturnal enuresis and four died at an early age from marasmus. Even those who had the nail and hair dystrophy had other abnormalities. One of these was an epileptic; one had cancer; four were feeble-minded, one had nocturnal enuresis.

This multiplicity of characteristics of degeneracy bears out the view that we are concerned in this family with a more fundamental disorder than that of an isolated abnormality of nails and hair. It would seem that there was present some organic defect that had widely distributed constitutional influences.

The well-known association of abnormalities of hair and of nails in hypothyroidism and the two fairly well defined cases of juvenile myxedema, and the reaction of one case to thyroid feeding, seems to warrant the conclusion that the fundamental disorder in this family was of the thyroid gland.

Cases Reported in the Literature.—There are in the literature a few other observations on families showing similar dystrophies of nails and hair. The earliest one of these and the most extensive in its abnormalities was that reported by Nicolle and Hallipré.¹ In this family of fifty-five known persons, thirty-six showed the dystrophies. In 1896 White² reported the study of a family in which there were fourteen members in four generations. Of these, seven showed the characteristic abnormalities. Eisenstaedt,³ in 1913, reported observations on a family of thirteen members which showed the occurrence of the dystrophies in five generations. Among eight members of the fourth generation, the abnormality was present in three. It is also probable that a contribution by Hoffman⁴ should be included in this connection. This reports a line of thirteen persons, eight of whom through four generations had shown abnormally short and scant eyebrows and dystrophies of the nails of the hands and feet. In this family there were several instances of thyroid disease. The patient had a large struma, as had her mother. The appearance of the mother

1. Nicollé and Hallipré: *Maladie familiale caractérisée par des alterations des cheveux et des ongles*, *Ann. de Derm. et Syph.*, 3d Ser. **6**:804, 1895.

2. White, Charles J.: *Dystrophia Unguium et Pilorum*, *Hereditaria*, *J. Cut. and Gen. Urin. Dis.* **14**:220, 1896.

3. Eisenstaedt, J. S.: *Three Cases of Family Dystrophies of Hair and Nails*, *J. A. M. A.* **60**:27 (Jan. 4) 1913.

4. Hoffman: *Ueber Verkümmern der Augenbrauen und der Nägel bei Thyroidosen*, *Arch. Derm. u. Syph.* **89**:381, 1908.

suggested hypothyroidism and Hoffman comments that "perhaps one might believe that a dysthyroidism was the predisposing cause for the described malformations."

The frequency of disorders of the nails or of the hair in hypothyroidism is shown in several analyses of large groups of cases of myxedema. In an analysis of 150 cases, Hun and Prudden⁵ found malformations of nails in 75 per cent. of cases studied, and in a later series Howard⁶ found nails abnormal in 86 per cent. of cases, and hair in 93 per cent. of those studied.

As to the heredity of hypothyroid disorders, there is a good deal of confirmatory evidence. In the previously cited analyses of large groups of cases, Hun and Prudden found a direct inheritance in 8 per cent. of cases studied, and Howard in 6.6 per cent.

It would be of interest to know to what extent some of the so-called neurotic disorders of the hair are brought about through the reaction of the thyroid gland to disorder of the sympathetic nervous system coming primarily from affective disturbances such as the sudden loss of hair or change in color following fright and nervous stress. There may be persons whose thyroid functioning is just sufficient to maintain normal health, and under stress this balance may be so disturbed as to produce definite pathologic conditions. In this group we have reported there were two persons who lacked the characteristic family dystrophies, who, following slight constitutional disturbances suffered a loss of hair and later regained normal conditions.

State Psychopathic Hospital, University of Michigan.

5. Hun and Prudden: Myxedema, *Am. J. M. Sc.* **96**: July and August, 1888.

6. Howard, C. P.: Myxedema: A Study, *J. A. M. A.* **48**:1226 (April 13) 1907; *ibid.* **48**:1403 (April 27) 1907.

CONGENITAL FACIAL PARALYSIS *

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LITERATURE ON THE SUBJECT

The significance of cases of congenital facial paralysis is still a question of considerable interest. For the last fifty years clinical observations with scanty pathologic reports have been recorded.

Anatômists, even such as Piersol,¹ who discusses anomalies under "Practical Considerations," do not mention congenital facial paralysis. This is true to a less extent of pathologists. Some of them do not mention congenital facial paralysis (Adami and Nicholls,² Bruning and Schwalbe,³ Monakow⁴). Others simply state that in gross mal-developments of the brain the cranial nerves also suffer (Ernst⁵); while others admit and discuss the possibility of congenital facial paralysis with reservations; for instance, Ballantyne,⁶ writing as follows: "Sometimes, as in a case about which I was consulted by Dr. Dickson of Lochgelly, 1899, the long persistence of the paralytic condition throws doubt upon the peripheral nature and traumatic origin of the palsy. Under these circumstances, it is reasonable to turn from an intranatal to an antenatal mode of origin of the nerve lesion. It may, then, be due to a lesion in the facial nuclei in the pons, or in the fibers connecting them with the cortical centers; but, of course, even

* Read at the meeting of the American Neurological Association, Atlantic City, N. J., June 16, 1919.

1. Piersol, G. A.: *Human Anatomy Including Structure and Development and Practical Considerations*, Philadelphia, J. B. Lippincott Co., 1907.

2. Adami, J. G., and Nicholls, A. G.: *The Principles of Pathology*, 2: Philadelphia, Lea & Febiger, 1909.

3. Bruning, H., and Schwalbe, E.: *Handbuch der Allgemeinen Pathologie und der Pathologischen Anatomie des Kindesalter*, 1: 1912.

4. Monakow, C.: *Gehirnpathologie*, Ed. 2, 1905.

5. Ernst, P.: *Missbildungen des Nervensystems*, in Schwalbe, E.: *Die Morphologie der Missbildungen des Menschen und der Tiere*, Part 3, No. 2, Section 2, Chapter II, pp. 100, 101, 251.

6. Ballantyne, J. W.: *Antenatal Pathology and Hygiene*, 1: New York, William Wood & Co., 1902, p. 47.

this central origin, or cerebral form, may be of intranatal origin, although it is unlikely."

Congenital facial paralysis has escaped the notice of Gould and Pyle,⁷ who discuss many rare and curious anomalies. It is not mentioned in *Nouvelle Iconographie de la Salpetriere*, 1877 to 1915.

Clinically, on the other hand, the existence of congenital facial paralysis has been definitely established, but many authors of systematic works do not mention it (Taylor,⁸ Burns, Cramer and Ziehen,⁹ Thompson,¹⁰ Turner¹¹). Others simply state the fact that congenital defect of the temporal bone causes facial paralysis (Jelliffe and Whitehall¹²), and only a few clinicians discuss cases reported in literature, for instance, Oppenheim,¹³ who states that the condition is associated with ocular palsies, deafness, defects of muscles and temporal bone, etc. The same is true of Vogt,¹⁴ whose remarks, however, are interesting, viz.; "In peripheral facial paralysis the whole side of the face is motionless; in central and in most of the bulbar (types) the superior branch retains its movement. In congenital cases the superior branch remains motionless, while the region about the mouth retains either complete or partial motility. When this difference between the upper and lower part of the face is great, it gives a characteristic facial expression: full, protruding lips which are in contrast with the mask-like, expressionless, sunken face above. This appearance of the face makes the condition often recognizable at the time of birth, and gives one the impression of an advanced atrophy or aplasia of muscles; also the skin over the muscles is usually smooth, peculiarly shiney, and colorless."

In periodical literature quite a number of cases have been reported. As early as 1898 an excellent paper was read before the American

7. Gould, G. M., and Pyle, W. L.: *Anomalies and Congenital Curiosities of Medicine*, Philadelphia, W. B. Saunders Co., 1897.

8. Taylor, E. W.: *Case Histories in Neurology*, 1912, in *Diseases of the Cerebral Nerves*. Osler's System of Medicine, Mod. Med. 5:471, 1915.

9. Bruns, L., Cramer, A., and Ziehen, T.: *Handbuch der Nervenkrankheiten im Kindesalter*, Berlin, 1912.

10. Thomson, H. C.: *Diseases of the Nervous System*, Ed. 2, New York, Funk & Wagnalls Co., 1915.

11. Turner, W. A.: *Diseases of the Cranial Nerves*, in *System of Medicine*, Allbutt & Rolleston, London 7:500, 1910.

12. Jelliffe, S. E., and White, W. A.: *Nervous and Mental Diseases*, Ed. 2, Philadelphia, Lea & Febiger, 1917, p. 291.

13. Oppenheim, H.: *Lehrbuch die Nervenkrankheiten*, Ed. 6, Berlin, S. Karger, 1913, p. 630.

14. Vogt, H.: *Infantile Beweglichkeitsdefekte im Bereich der Hirnnerven*, *Handbuch der Neurologie* (M. Lewandowsky), Berlin, 1911, p. 270.

Neurological Association by Dr. H. C. Thomas,¹⁵ based on the study of two brothers. Dr. Thomas reviewed the literature in detail and, concerning his cases, summarized as follows: "That the muscle defect in these cases is a congenital one, I think cannot be doubted. In the first place, the mother is quite sure that the defect was present when the children were born, although she cannot state that she noticed the deformity immediately after birth. The fact that the malformations occurred in two members of the same family, and that it is bilateral, speaks for its congenital origin, and the occurrence of other faults in development, the misshaped ears and possibly the deafness, lends added weight to this view. The character of the paralysis itself is quite similar to that which is found in the other congenital cases."

In recent years communications on this subject have appeared from Langdon,¹⁶ Hutchinson,¹⁷ Rainy and Fowler,¹⁸ Batten,¹⁹ Harris,²⁰ Neurath,²¹ and others. Heller,²² summarized in his Paris thesis the relations of congenital facial paralysis to agenesis of the temporal bone. His conclusions are as follows: "There is a special variety of congenital facial paralysis, which is caused by agenesis of the temporal bone. It is due to arrested development of the petrous portion and portions of the auditory apparatus."

"It may be complete, with total loss of voluntary muscular contractions and of electrical reaction, or partial, with preservation of the function and characteristics of the facial nerve."

To this type belong the cases of Harris²⁰ and Neuenborn.²³

15. Thomas, H. M.: Congenital Facial Paralysis, *J. Nerv. & Ment. Dis.* **25**:571, 1898.

16. Langdon, F. M.: Facial Paralysis, Congenital, Unilateral and of Unique Distribution, *J. Nerv. & Ment. Dis.* **26**:593, 1899.

17. Hutchinson, Sir J.: Persisting Paralysis of the Muscles Supplied by the Facial Nerve, *Arch. Surg.* **2**:20, 1900.

18. Rainy, H., and Fowler, J. F.: Congenital Facial Diplegia Due to Nuclear Lesion, *Rev. Neurol. & Psychiat.*, 1903, p. 149.

19. Batten, F. E.: Congenital Affection of Sixth, Seventh and Twelfth Cranial Nuclei, *Brain* **28**:359, 1905.

20. Harris, H. E.: Congenital Absence of the Left Ear with Facial Paralysis, *Brit. J. Child. Dis.* **1**:502, 1904.

21. Neurath, R.: Zur Frage der Angeborenen Funktionsdefekte in Gebiete der Hirnnerven, *München. med. Wchnschr.* **54**:1224, 1907.

22. Heller, A.: Contribution a l'Etude des Paralysies Faciales Congenitales, par Agénésie du Rocher. *Theses de Paris*, 1903.

23. Neuenborn, R.: Rudimentaer Entwickelte Missbildete Ohrmuschel mit Congenitaler Einseitiger Facialislahmung Infolge Hypoplasie des Nerven, *Arch. f. Ohren* **63**:58, 1904.

Recently the maldevelopments of the ear have been studied by Marx,²⁴ who wrote a profusely illustrated chapter in Schwalbe's *Morphologie der Missbildungen*, in which he touches on congenital facial paralysis. He believes this condition is mostly secondary to anatomic defects of the middle ear, but in some instances due to primary hypoplasia or agenesis of the facial nerve. This, according to Neurath,²¹ practically summarizes all that is known about the pathology of congenital facial paralysis, and we may add that it is frequently associated with many other congenital anomalies. To correlate these anomalies pathogenically is often very difficult. He says: "The existence of congenital nuclear aplasia, although rare, has been definitely established" (Zappert²⁵). Supporting this statement is the instance by Rainy and Fowler,¹⁸ who found that "the facial nerve on both sides showed very marked degeneration, alike in the ascending part of the root, in the fasciculus teres, and in the emergent portions. Degenerated fibers could also be clearly traced passing through the healthy sixth muscle in the manner described many years ago by Gowers and von Gudden." The facial nuclei were also found to be pathologic.

"In a number of cases, especially in paralysis in the region of the eye muscles and of the facial nerve, a peripheral affection (muscle, nerve) is proven" (Zappert²⁵).

REPORT OF CASE

Physical Examination.—W. B., female, aged 11, was sent to Dr. Fry for examination by Dr. R. N. Crews, Fulton, Mo., June 27, 1918.

The facial palsy of both sides is evident at a glance. Only a few minutes' inspection is necessary to reveal that it is complete and total. There is no motility whatever, except a slight drawing down at the corners of the mouth; this is evidently accomplished by the platysmata and is a little more pronounced on the left side. She is a well nourished, healthy child, with good complexion and hair, yet the absence of animation in her countenance is striking. When instructed to close her eyes, the mask effect is complete, the clear sclera presenting through a palpebral fissure a little over a quarter inch wide on each side. The skin is clear and smooth over the whole face, and only slightly thinned over the forehead and over the malars and nose. There is no corrugation, with the exception of a slight corrugation about the eyes and mouth. The upper lip is thin and slightly pursed, and the lower slightly everted. There is very little drooling and only when speaking or eating.

There are no lateral movements of either eyeball. There is a good range of vertical movement, and it is the same for each eye. The downward range is greater and seems quite full; the upper range is possibly somewhat limited.

24. Marx, H.: Die Missbildungen des Ohres, in Schwalbe, E.: Die Morphologie der Missbildungen des Menschen und der Tiere, 3: Part 3, No. 2, Section 2, p. 595.

25. Zappert, J.: Ueber Infantilen Kernschwund Ergebnisse der Inneren Medicine und Kinderheilkunde 5:305, 1910.

There are no oblique movements, or, if any, so slight as to be questionable. The pupils are equal, and react well to light and to accommodation. The ciliary function is intact. In making the above observation, we had the valuable assistance of Dr. Joseph W. Charles, who joined us in examining the eyes.

There is no lateral movement of the mandible. The members of her family seem quite aware of this, and her father, who accompanied her, offered to

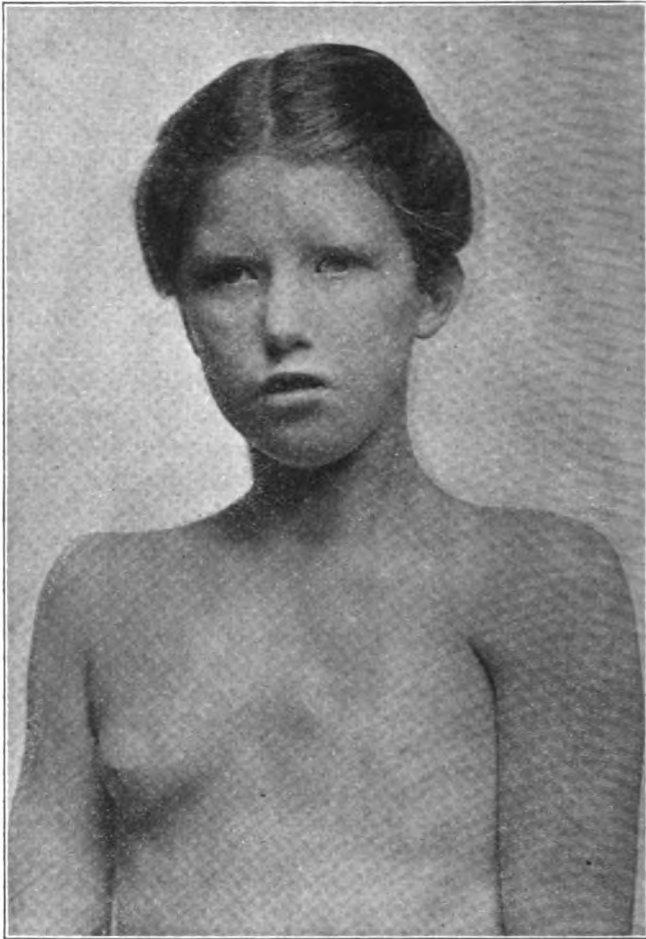


Fig. 1.—A girl, aged 11, in whom there is a complete absence of mobility in the distribution of the seventh nerve on both sides of the face. There is also an absence of all lateral movements of the eyeballs, mandible and tongue, and a teratologic absence of the left breast.

demonstrate her manner of masticating by furnishing her some gum to chew. The range of vertical movement of the jaw is possibly somewhat limited, but not much, and the strength of contraction seemed full enough.

The tongue is atrophied and corrugated; somewhat more so on the left. Its movements are limited and wobbly.

The range of movement in the soft palate and uvula is quite limited. It also has a corrugated appearance, more so on the left. The whole faucial surface seemed to be exceedingly sensitive, making the examination of it difficult, although the patient tried to acquiesce. There seems, however, to be little, if any, difficulty in deglutition. There are no motor defects in other portions of her body. The reflexes generally present no abnormalities.

Sensibility everywhere seems intact and well up to normal standard, including the special senses and general sensibility.

Mental Examination.—In intelligence and character this little girl seems quite up to the average of her age. Her speech is, of course, very defective; in fact even to her father it is almost unintelligible. However, her brother and sister and her teacher understand her "lingo" very well. She does not use standard sign language, and, although she writes very well indeed for one of her age, she does not converse in this way to any extent; nor does she use much pantomime beyond some coy little movement of her shoulders and hands for expressional purposes.

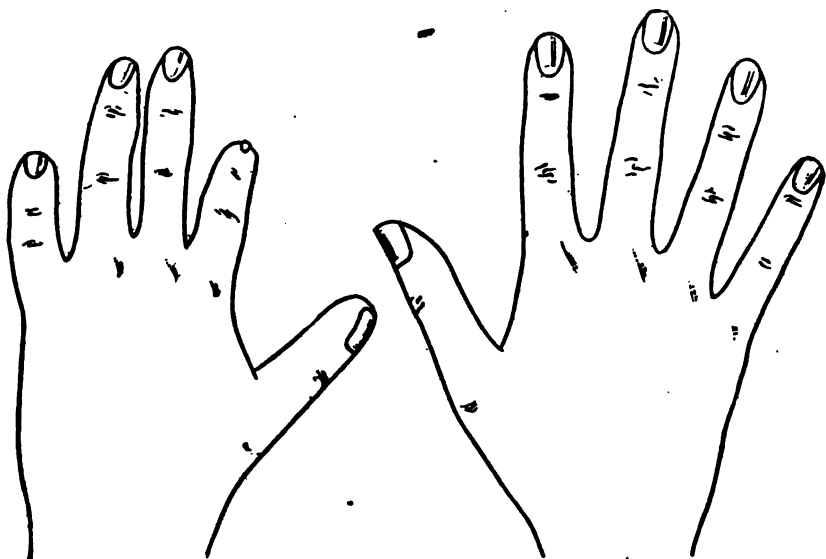


Fig. 2.—This figure shows the relative size of the right and left hands and the teratologic deformity of the left.

Although her face is so utterly expressionless (the more so on account of the limited eye movements), and her speech very defective, her demeanor is otherwise so alert that one is quickly assured of her intelligence. In the record are specimens of her handwriting and figuring that are very creditable.

History.—The parents have always been aware that the defects in the child were congenital; they have also believed they were hereditary, for there is in the family a tradition at least, that all the members of the mother's family have a webbing of the second and third toes, and a report that the mother's father had a withered arm from birth and that two of the mother's brothers had congenital deformities. The details, however, were not obtainable.

The mother and father of our subject and their other children present no abnormal features. They are all "healthy and well."

At the birth of W. B. (our subject) the delivery was precipitous, and the mother was unattended by a physician for an hour after the child was born, but there is no record of any untoward result to the child. When she was a year old her health seemed perfect. In her second year she had pneumonia, and for several years thereafter was rather delicate, especially suffering with conjunctivitis, which resisted ordinary methods of treatment. By the time she was 8, however, she was well and strong. In her ninth year her tonsils and adenoids were removed. Since she has been perfectly well, "full of life, jolly, and an excellent student."

The left mamma is entirely absent, the merest semblance of a nipple persisting. She seems to have fair strength in the pectoral muscles of this side, but the flat, attenuated appearance of the breast in contrast with the other side, gives the right side the appearance of being over-developed for her age.

The appearance of the left hand is so evidently teratologic, that only a brief description is necessary. The index and little finger are so short as to be quite out of proportion to the other digits. The whole hand is considerably smaller than the right. There is no atrophy, and she uses it very well, but not so freely as the right. The muscles of the arm are all present and proportionate, although the member is somewhat smaller than the right, both in length and general circumference.

We shall not in this connection attempt to discuss the teratologic bearings of the case, more than to suggest that the deficiencies here present are of the type due to germinal defect as distinct from the teratologic types caused by mechanical process in utero, as, for example, club-foot, cyclopia, spina bifida, etc.

PROBLEMS IN THE DIAGNOSIS AND TREATMENT OF INJURIES TO THE PERIPHERAL NERVES

THE OUTLOOK FOR THE FUTURE *

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This paper, based on observations made on 170 patients with injuries of the peripheral nerves whom we have studied at U. S. Army General Hospital No. 1, deals only with some problems which we have encountered and with the means and methods by which we have attempted to solve them. The paper concerns itself somewhat with the diagnosis of the injuries, but chiefly with the general conditions on which successful treatment must depend. We have not allowed ourselves to draw any conclusions as to the final results of the methods of treatment used as several years must elapse before conclusive data can be forthcoming. We shall, in what follows, speak of the significance of some symptoms and signs of nerve injury, of some aspects of the finer anatomic structure of the peripheral nerves, and of the anatomic basis and guiding principles for the technic of nerve suture and nerve grafting.

I. THE SIGNIFICANCE OF SOME NEUROLOGICAL SYMPTOMS AND SIGNS OBSERVED IN NERVE INJURIES

1. *The "Tinel" Sign.*—Much has been written concerning the importance of the tingling that can be observed when the skin is tapped along the course of a peripheral nerve. According to Tinel, tapping distally to the site of the nerve injury causes tingling referred to the area of distribution of the nerve only when the nerve in question is regenerating. On the other hand, tingling cannot be observed when a peripheral (mixed) nerve has been completely divided. Tinel claims also that in a regenerating nerve the tingling can be obtained on tapping at the points along the nerve more and more toward the periphery corresponding to the gradual downgrowth of the axis cylinders.

We are not yet in a position to deny or affirm that formication obtained by tapping on the nerve distally to the point of injury occurs

* Read at the meeting of the American Neurological Association, Atlantic City, N. J., June 16, 1919.

only, or invariably, when sensory fibers are regenerating. The value of the sign from the practical diagnostic standpoint is doubtful. We have in not a few cases observed this distal formication in patients in whom operation later showed a defect in the nerve with no discoverable (macroscopic) fibers bridging the gap. But even though the sign definitely indicated regeneration of sensory fibers, this does not necessarily mean that regeneration of motor fibers has occurred or will occur. Practically the necessity for surgical treatment was decided in every one of our patients by the behavior of the motor fibers. If motor fibers were not regenerating in sufficient numbers by the sixth to the eighth months after the injury, whether the sensory fibers were functioning well or not, it was assumed that a serious impediment was obstructing their progress, and exploration was advised. In all except two of the cases of this series (to be mentioned below) in which operation was advised and carried out, either complete severance of the nerve was found, or such involvement in scar tissue or fraying of the nerve, as made spontaneous progress impossible.

2. *The Skin Area Over Which a Given Sensory Nerve is Distributed Varies Considerably in Size in Different Individuals.*—Thus, if sensation remains normal in the superficial radial area in patients with musculospiral injuries, one must not be oversanguine in hoping therefore to find an incomplete severance of the nerve. In some persons the area supplied by the superficial radial is, like that of the anterior tibial nerve in the foot, only a penny-sized spot. Similarly, in one of our patients with a sciatic injury, there was merely reduction of sensibility in the musculocutaneous area of the external popliteal nerve, and we had expected accordingly to find some sound fibers passing through the damaged portion of the sciatic nerve. But the operation showed the upper stump of the external popliteal bundle of the sciatic disconnected from the distal stump and driven back nearly at a right angle to its normal position. The area was presumably supplied by overlapping fibers from neighboring nerves.

In making inferences based on sensory findings one must, therefore, keep in mind these individual variations as to distribution, and that the overlapping fibers from contiguous nerves gain in acuity of perception as the patient is forced to depend on them. Whether or not the overlapping fibers may actually progress farther into a defective area as time elapses after the injury, we are unable to say from any experience gained in the present study.

3. *Symptoms and Changes in Nerves Due to Ischemia.*—In four of our patients, in addition to the injury to the nerve, the main blood vessels of the limb had been severed when the wound was inflicted, or closed by the subsequent contraction of scar tissue. In many the

nutrient arteries to the nerves had been destroyed. And in most patients whose wounds were in nerves that lay adjacent to large blood vessels, the blood supply of the limb distal to the wound was reduced by pressure on the vessels.

Muscles and nerves respond promptly to quantitative or qualitative changes in the blood because of the complexity of the biochemical changes that occur in them. In the case of nerve fibers, their attenuated form and their dependence on the distant cell bodies for trophic control cause them to suffer doubly under the conditions mentioned above. These patients showed symptoms that were not confined to the distribution of the nerves that had been wounded. A general reduction of sensation was found throughout the limb. The muscles controlled by other nerves were weak and atrophic. Coldness of the skin, subjective and objective, and cyanosis in some cases, were present.

Because of the frequency of this factor of ischemia from pressure on blood vessels, we became more confident as our experience progressed in operating to free nerves and vessels from pressure. For the removal of scar pressure not only liberates the fragile axons and restores better blood supply at the point of injury, but it improves the nutrition of the tissues below the wound which had labored under the double disadvantage of insufficient blood and poor trophic stimulation from the spinal centers.

4. *Intraneural Blocking of Axons Without Gross Injury to the Nerve.*—This condition was found in two of our patients. In one of them, following a compound fracture of the bones in the lower third of the leg, in October, 1918, there were, in May, 1919, complete sensory loss in the distribution of the posterior tibial nerve and complete loss of power in the intrinsic muscles of the sole without any signs of regeneration. We had expected to find a complete severed posterior tibial nerve or one badly compressed by scar or callus, but the incision revealed a nerve normal in appearance with only one slight adhesion and without any involvement in scar tissue.

In the other patient there were two gunshot wounds in the arm in the course of the musculospiral nerve, dating from November, 1918. There was a large scar in the axilla and another on the lower half of the arm on its outer and posterior aspect. Pressure on the scars gave pain. Palpation gave no information. The triceps was weak, the supinators and all of the extensors were without function, there was complete reaction of degeneration in the nerve and muscles; there was total anesthesia of the radial cutaneous area. In this case the operation in May, 1919, showed that all the wounds were superficial and the tissues around the nerve in both locations appeared normal, as did the nerve itself.

In each of these patients, therefore, a wound had been received along the course of a nerve, and six to seven months afterward no function had returned in the nerve below the point of injury. Yet there was no change in the nerve trunk, visible to the naked eye, adequate to explain the interruption of function. It appears from these two cases that a trauma which causes no macroscopic change in a nerve trunk can sever its axis cylinders within the epineurium, or at least inhibit their function for six or seven months. We did not have opportunity to study a nerve microscopically to learn what minute changes had occurred. Aside from direct inspection of the nerve after cautious exploratory incision, we know of no diagnostic method by which this intraneural interruption of axons could be differentiated from grosser injuries.

5. *Substitute Movements.*—In studying the voluntary movements of patients the inference is ordinarily justifiable that if a patient is able to pronate his forearm, his pronator muscles and therefore all or part of his median nerve must be sound; if he is able to extend his wrist, that the extensores carpi, and therefore his musculospiral nerve are functioning. Yet care is necessary in making these inferences, for patients in whom the median nerve has been divided with paralysis of the pronator muscles, learn in the course of time to use other muscles to imitate the movement of pronation. Similar substitute movements were observed in other nerve palsies, a few of which we mention (see Figs. 1, 2, 3, 4).

(a) An imitation of flexion or extension produced by a pull in the wrong direction by the normal antagonist muscles, and an immediate rebound of the joint. This rebound is easily mistaken for a positive motion, and in these cases it appeared that a muscle actually paralyzed was contracting. Thus, in several patients with complete external popliteal paralysis some dorsal extension of the toes seemed possible. When the toes were carefully watched, the dorsal extension movement was seen to be simply an elastic rebound after a quick and very slight plantar flexion.

(b) A paralyzed and fibrosed muscle whose tendon extends over two joints can be pulled on by the action of its antagonists, and thus a movement can be secured which resembles the one formerly caused by the paralyzed muscle. A frequent example of this was the extension of the wrist by patients with musculospiral paralysis through the device of flexing the fingers and thus drawing the extensor tendons taut over the knuckles. This tension on the extensor muscles of the fingers extends the wrist.

(c) Patients with complete median nerve paralysis may be able to pronate the forearm by contracting the extensors of the wrist so as to turn the hand through about 90 degrees and then allow the flexed hand to drop by gravity into the prone position. This is easily mistaken for normal pronation.

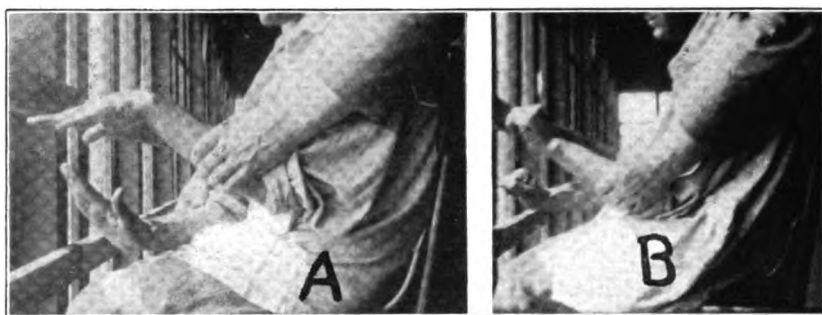


Fig. 1.—Paralysis of muscles dependent on left musculospiral nerve. In picture A, the patient is trying to extend his wrists while holding the fingers straight. He succeeds in the right hand, fails in the left. In picture B he has extended his left wrist by flexing the fingers. (He unconsciously imitated this movement in the right hand.)



Fig. 2.—Paralysis of muscles supplied by the right median and ulnar nerves; marked contracture of flexor muscles of fingers. In picture B the patient is attempting to flex his fingers beyond the position presented. Picture A shows the amount of flexion secured in the fingers by extending the wrist. It enables him to grasp and hold small objects like a lead pencil.

(d) It is believed by some authorities that extension of the second and third phalanges of the fingers is caused only by the interossei (ulnar nerve). But the musculospiral extensors of the fingers also act on these distal joints, as well as on the proximal ones. Thus in ulnar nerve injuries one must not argue that some ulnar function persists simply because extension of the terminal phalanges is possible.



Fig. 3.—Right median nerve injury. Paresis of long flexors of digits. In picture A all fingers are extended. The patient is about to attempt to flex them. In picture B he has flexed his left fingers without disturbing the wrist. In the right hand he flexed the fingers only by extending the wrist.

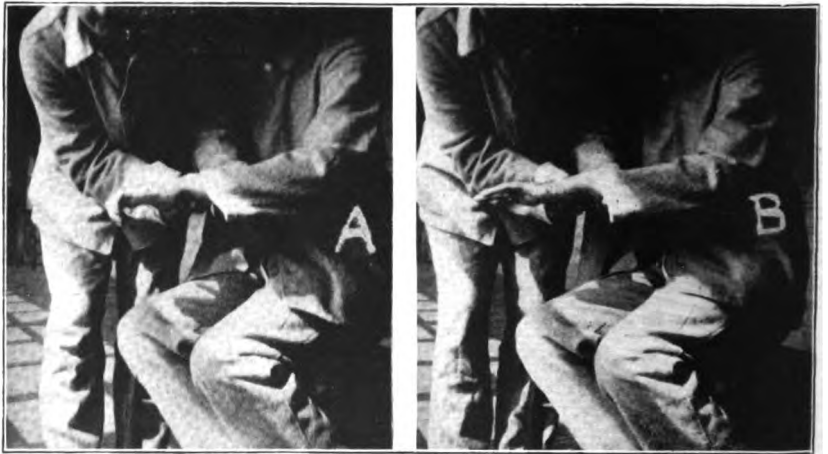


Fig. 4.—Paralysis of interossei muscles following complete severance of left ulnar nerve. Picture A shows the proximal phalanges held in extension, the second and third phalanges flexed. In picture B the patient has extended the second and third phalanges in spite of the paralysis of the interossei muscles. This was done presumably by the extensor communis digitorum.

II. THE FINER ANATOMICAL STRUCTURE OF THE PERIPHERAL NERVES
AS THE ANATOMICAL BASIS FOR NERVE SUTURE; GUIDING
PRINCIPLES FOR THE TECHNIC OF NERVE
SUTURE AND NERVE GRAFTING

From the beginning of a peripheral nerve operation to its end a perfect technic is necessary. The exposure of the nerve by an incision which will cause the minimum amount of injury to muscles, the freeing of the ends of a divided nerve and the excision of the surrounding scar tissue with the gentlest handling of the nerve, the perfect control of bleeding so as to have a dry wound, the accurate sectioning of nerve bulbs until good nerve fibers are exposed, the proper approximation of the nerve ends without tension, and the properly applied and tightened suture—these and many other details are of great importance. One minute fault in technic, and the chances for a good nerve regeneration are much diminished, no matter how skilfully all other manipulations may have been accomplished.

The ideal technic in nerve suture toward which surgery, therefore, must develop is one that will approximate the proximal stumps of as many as possible of the severed axons to the original glial channels of the peripheral stump through which they formerly reached their destinations; and this must be done without further injuring the delicate axons, and without adding obstacles to their passage into the appropriate channels.

The technic described is the nearest approximation to this ideal we could devise.

1. *The Handling of Nerves During Operation.*—The surgeon cannot be too careful in his handling of the exposed nerves. Great care and gentleness are necessary in all manipulations, and the rule should be to handle the nerves as little as possible. While it is permissible to grasp scar tissue around a nerve with forceps, the nerve should never be held with a steel instrument. One well known operator uses glass hooks for picking up nerves during operation, not because they are softer or smoother than steel, but “for their psychic effect on the operator and assistants.” If a nerve is seized by a tissue forceps the fibers thus compressed may be, and no doubt usually are, immediately destroyed at that point. Thus to roughly seize the innervating nerve that goes to the *tibialis anticus* will cripple the patient the same as if the tendon of that muscle had been cut at the ankle. To nip the ulnar nerve in the arm in a forceps may interrupt the bundle of fibers that control the *interossei*, and destroy all the finer hand movements.

The nutrition of a nerve is imperiled by dissecting it out of the areolar tissue that surrounds it and supplies it with blood and lymph. There is a strong temptation when operating to “clean up” the nerve

trunk as if for a museum dissection. No further dissection is warranted during operation than that which is necessitated to identify the nerve, and to free it from scar pressure. Dissection for identification is required in inverse proportion to the operator's anatomic knowledge.

Furthermore, the lower end of a divided nerve should always be exposed and freed first, because it is the degenerated end. The central end should be exposed for as short a time as possible and should be handled with extreme care. Strong traction should never be made on it in the effort to approximate the divided ends of the nerve. We very emphatically advise against the use of manual methods or of appliances to make rapid or gradual traction on the ends of a nerve. If there is difficulty in drawing together the two stumps, the operator may be tempted to push a finger or blunt instrument along the course of the nerve above and below in order to free it from its surroundings. This dissection breaks away the vascular connections of the nerve, and endangers innervating branches to neighboring muscles. After all inflammatory adhesions and scar bonds have been released, it is doubtful whether the areolar tissue around a nerve restrains it sufficiently to make this procedure necessary.

2. *Salvaging of Nerve Fibers.*—When any bundles of good nerve fibers could be found running through a scar mass, they were carefully dissected out by longitudinal strokes of a knife or blunt dissector, and if possible, saved even though the remainder of the cross section had to be resected and sutured (see Figures 5, 6, 7). But if such a bundle of salvaged nerve fibers lay near the center of the cross section involved it had to be resected to prevent interference with the proper coaptation of the whole nerve.

In many cases where a number of such fasciculi could be discovered in a tangle of fibroneuroma, especially if the record showed that some motor impulses were permeating the obstruction, it was difficult to decide what course to pursue. If extreme conservatism prompted to simply free the nerve from external scar tissue and leave the fibroneuroma unresected, there was the counterbalancing fear that no other fibers but these would ever penetrate the mass, and that even these would sooner or later be compressed and destroyed. On the other hand, there was the fear that when resection and suture had been done, the union would prove unsuccessful and the little function that persisted would thus be lost.

The adage, "leave well enough alone," can always be answered by "good is the worst enemy of best." There are few situations in surgery in which rule-of-thumb is less efficacious, and in which the patient's welfare depends so completely on the sound judgment of the operator, as in this decision whether or not to resect a scar-entangled or frayed nerve.

3. *The Excision of the Bulbous Enlargement or of End Bulbs.*—The bulb or end bulbs should be divided transversely with a sharp scalpel in successive sections until normal nerve bundles without scar tissue around them are exposed. As the upper end of an injured nerve is often swollen, perfectly good funiculi may present edematous or

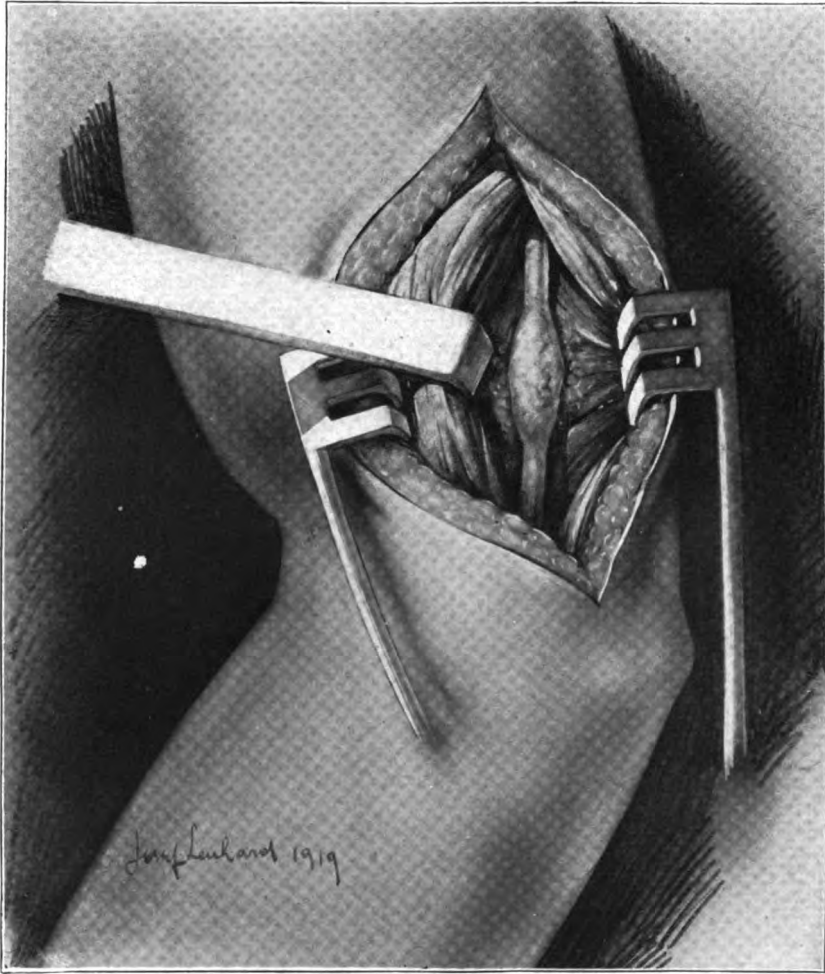


Fig. 5.—Injury of musculospiral nerve by shrapnel; bulb in continuity.

glairy surfaces. There is an interesting and easily understandable difference between the central and peripheral end bulbs (see Figure 8). When the peripheral end bulb is being sectioned the successive cross sections present the appearance of smooth, shiny scar tissue until one section is reached which contains the ends of many normal funiculi

without any scar tissue around them. When, on the other hand, the successive sections of the central bulb are examined, the transition from scar to normal is much more gradual. In successive sections there are an increasing number of good funiculi until an altogether normal transverse section is exposed.

4. *Some Features of the Gross and Minute Anatomy of the Peripheral Nerves, Nerve Pattern, the Prevention of Rotation of the Nerve Ends and Distortion of the Nerve Pattern.*—As we have stated above,

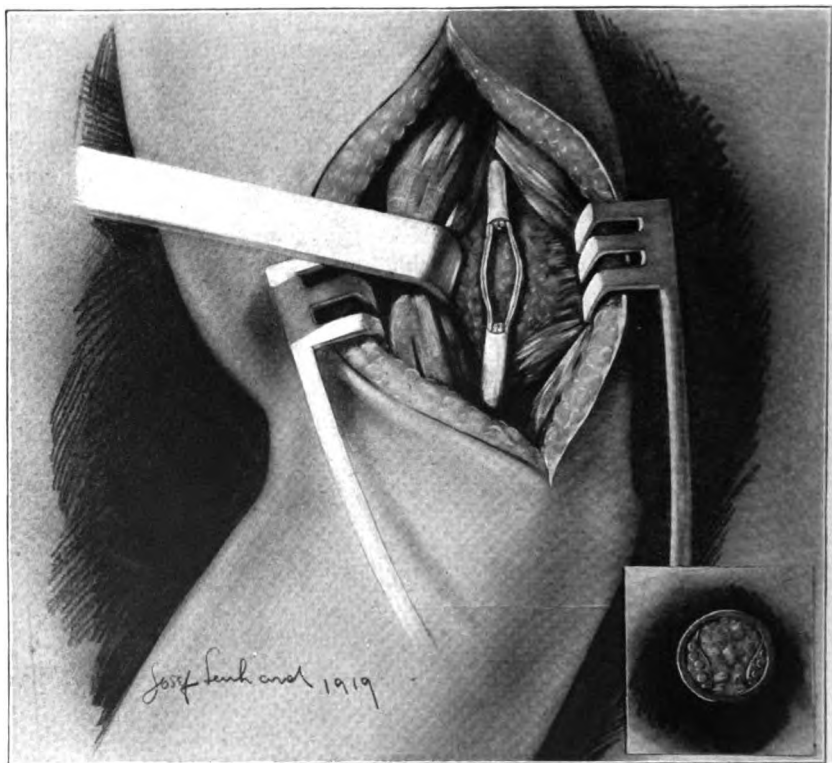


Fig. 6.—Same as Figure 5. The scar tissue has been excised and bundles of good nerve fibers preserved. The insert shows a theoretical transverse section of the bulb.

the ideal apposition of the ends of a nerve would be one in which the cut end of each divided funiculus was placed exactly opposite to its mate. While this ideal cannot be attained, the effort should be made to approach it as nearly as possible. For this purpose, a good understanding of the arrangement of funiculi in the different nerves, and in the different parts of each nerve, is indispensable.

It is remarkable how little is known of this subject. The investigations of Stoffel, Compton, Langley and Hashimoto and others have made it probable that each nerve has a definite nerve pattern, although the pattern may and does vary in different parts of the same nerve. Ideal peripheral nerve surgery can only be done when we have a very complete knowledge of the arrangement of funiculi and fibers in the peripheral nerves. We have made a large number of observations in our nerve operations and have carefully noted the arrangement of funiculi in the cross sections of the nerves. In the majority of

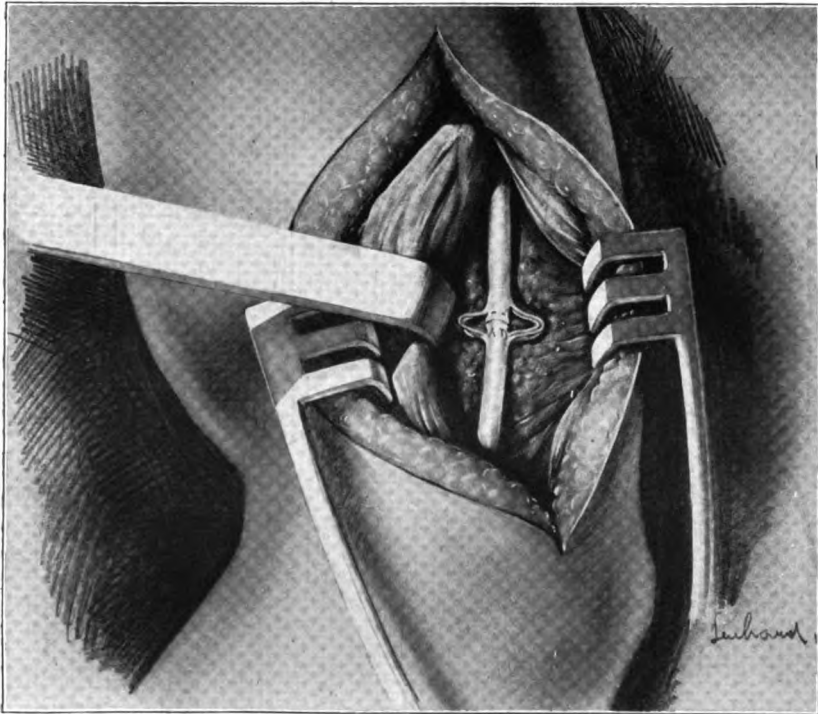


Fig. 7.—Same case as Figures 5 and 6; the suture completed.

instances the nerve patterns in the peripheral and central ends, visible to the naked eye, were either identical or were very similar. An entirely different arrangement in each end was observed in a surprisingly small number of the patients.

This subject is one of such great importance that Dr. Henry A. Riley and one of us (Elsberg) have been investigating it on human cadavers. The studies are far from complete, but they show that in each nerve there is, at different levels, a definite grouping of funiculi, easily recognizable with the naked eye. In many instances these group-

ings are so regular that, by a proper nerve suture, the end of each divided funiculus can be brought into approximate apposition to the corresponding end, and rotation of the ends of the divided nerve and distortion of the nerve pattern can be prevented.

We can obtain additional knowledge of nerve pattern by the electrical stimulation of the ends of a recently divided nerve, and one

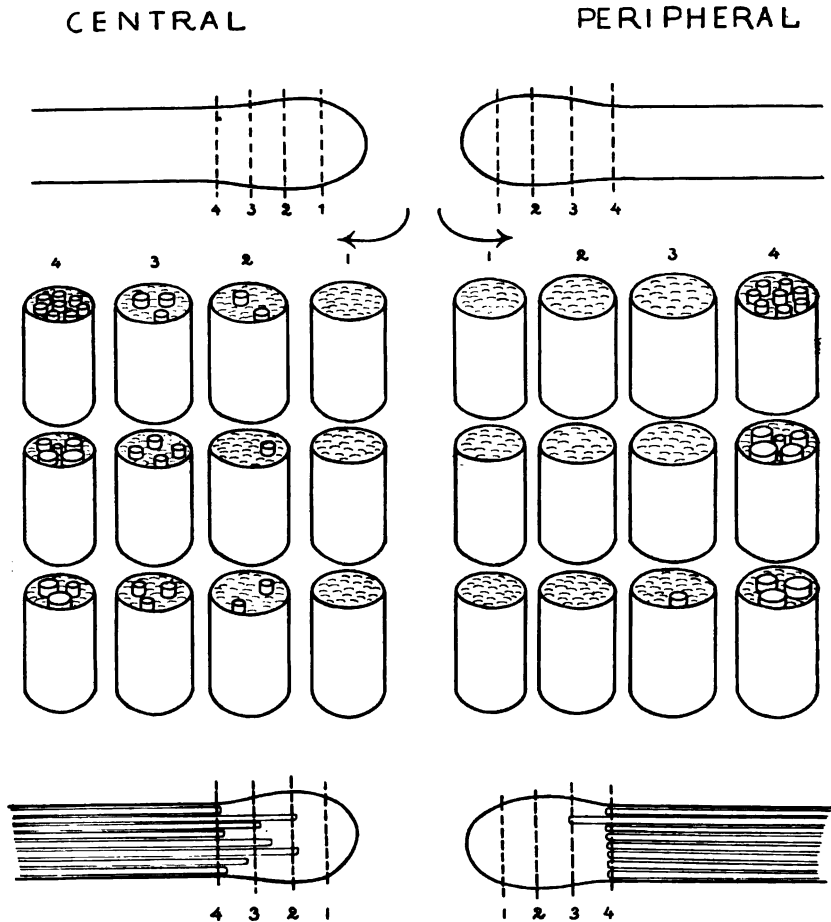


Fig. 8.—The difference between peripheral and central end bulbs.

of us (Elsberg) is also investigating this subject on the nerves of freshly amputated limbs. The investigations are not complete, but a number of definite records have been obtained which we believe are of great importance. A report on these studies will be published later.

The nerves, as regards their funicular pattern, may be divided into two main groups: (a) Those in which the number of funiculi is small,

but the funiculi are of large size—the type with large funiculi—and (b) those in which the number of funiculi is large, but the funiculi are of small size—the type with small funiculi. The musculospiral and external popliteal nerves belong to the group with large funiculi; the median, ulnar and internal popliteal belong to the group with small funiculi.²

Regeneration after suture is more rapid and more complete in the musculospiral and external popliteal nerves which we include in the group with large funiculi than in the median, ulnar or internal popliteal

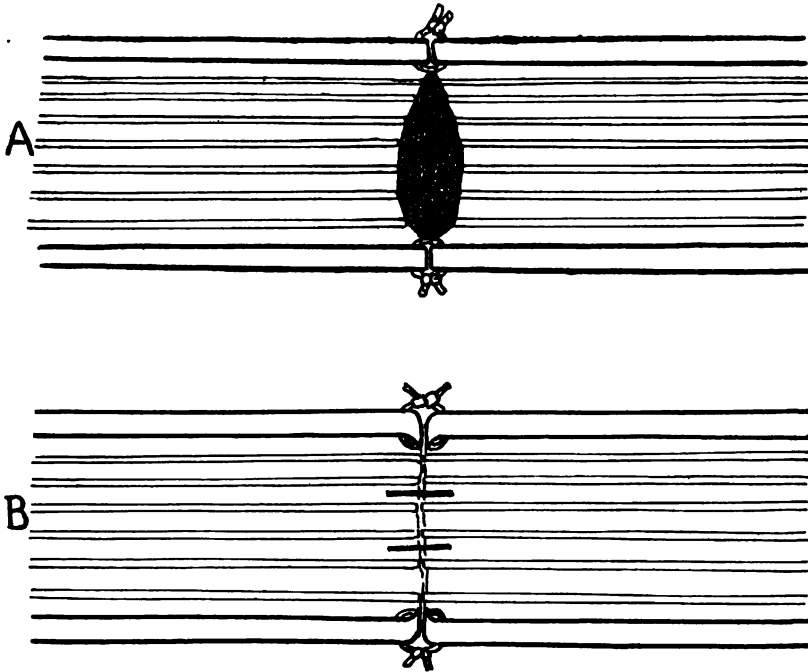


Fig. 9.—A, the condition that may exist when the ends of a divided nerve are united only by epineurial stitches. B, demonstrating more perfect approximation by a combination of perineurial and epineurial stitches (diagrammatic).

nerves with numerous small funiculi. This funicular arrangement may have an important bearing on regeneration after suture, for it is obvious that a given amount of scar tissue is more apt to impede the downgrowth of, and to compress the axis cylinders in a small delicate bundle than in a larger one.

There is another aspect of the subject which is of interest. Severe neuralgias and the so-called causalgia occur most often in the median

2. As we have stated elsewhere, there are variations in the pattern in different parts of the same nerve. This subject will be considered in detail in a separate paper.

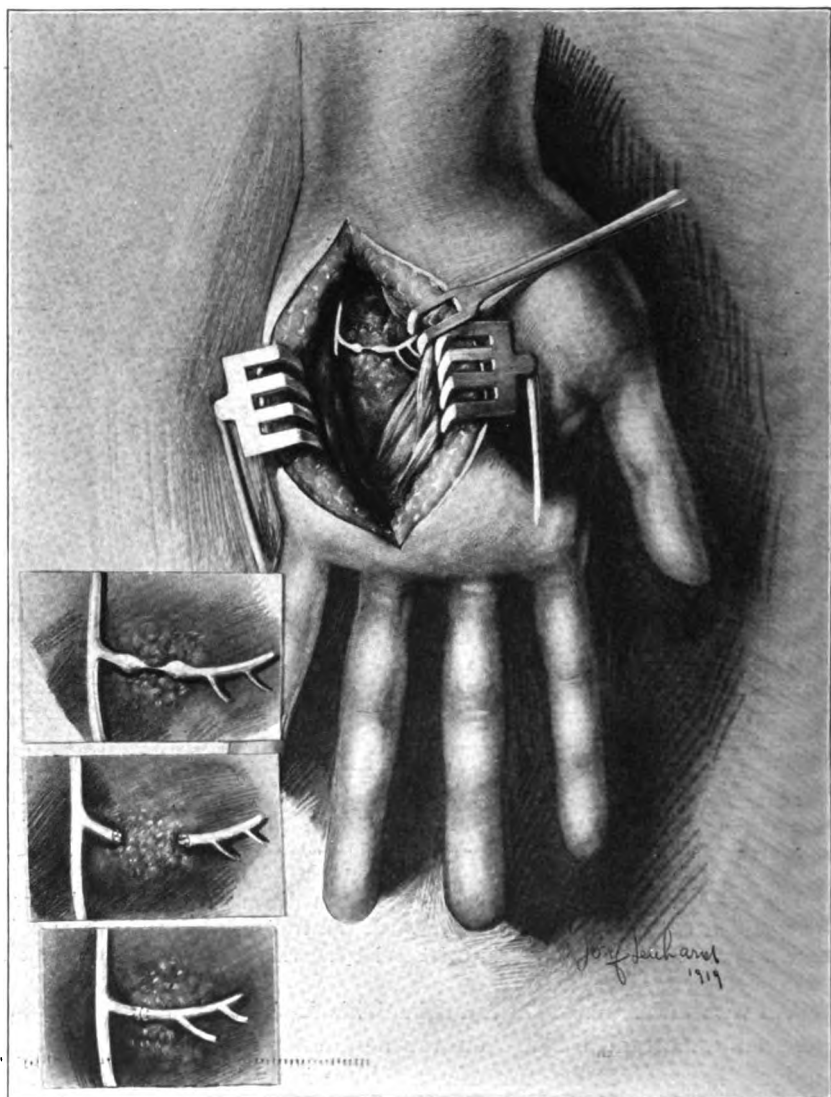


Fig. 10.—Division of deep palmar branch of ulnar nerve by machine-gun bullet. Resection of end bulbs and end to end suture. (The inserts show the successive stages of the operation.)

and internal popliteal nerves with numerous small funiculi. The electrical studies of one of us seem to indicate that in very many instances (perhaps in all) the sensory fibers occupy the most central part of each funiculus. May it not be that in inflammatory processes in the perineurium, an irritation of sensory fibers is much more apt to occur in a nerve with small funiculi than in one with large ones? We hope that the studies we are making will soon permit a definite answer to this question.

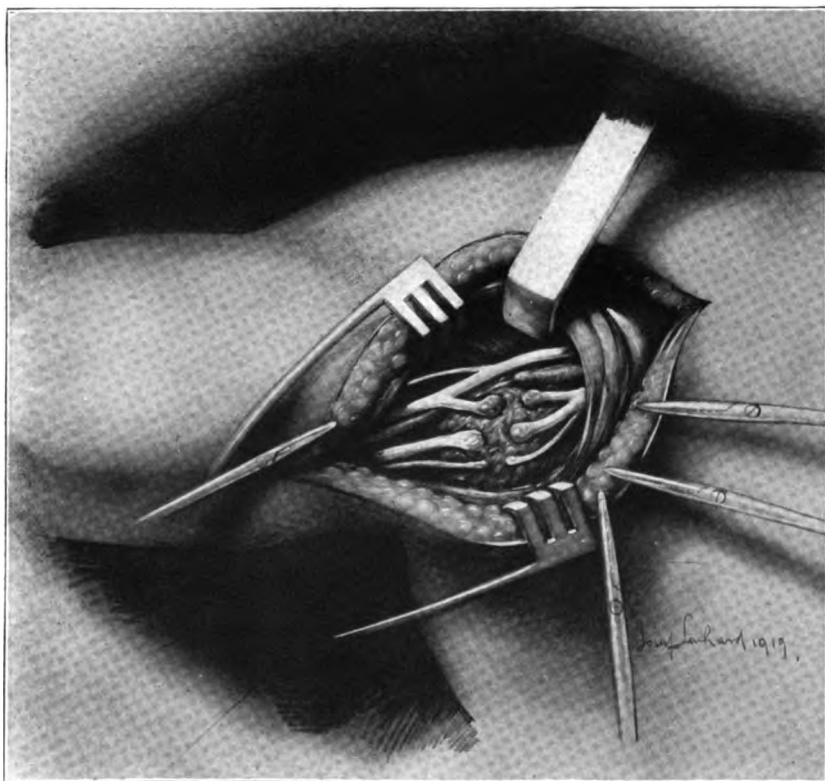


Fig. 11.—Shrapnel injury involving the inner head of the median, the ulnar, the internal cutaneous nerves, and the brachial artery. The end bulbs exposed.

5. *Approximation of the Divided Ends of a Nerve.*—This should always be made without tension. In the majority of instances, this can be accomplished by freeing the nerves—especially the peripheral part—for a considerable distance. In this procedure, however, due consideration should be given to the location of branches and of nourishing blood vessels so that important motor and sensory branches are not injured. The nerve ends can often be approximated without

tension by flexion of adjoining joints (flexion at wrist for injury of ulnar or median nerves in forearm; flexion at elbow for median and musculospiral in arm and forearm; extension at elbow and adduction of arm for ulnar nerve in arm and forearm; plantar flexion at ankle and flexion at knee for posterior tibial; flexion at knee and extension

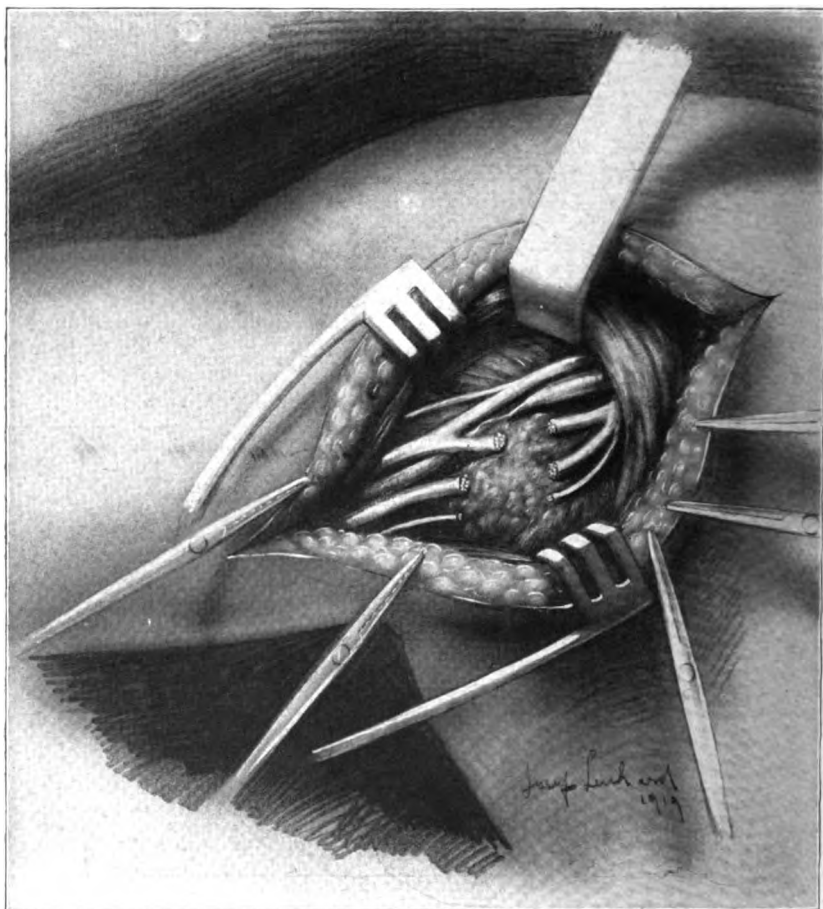


Fig. 12.—The end bulbs resected, showing the funicular pattern of the ends of the nerves.

at hip for sciatic, etc.). Transplantation of the ulnar nerve to the front of the internal epicondyle is often necessary, but in this transplantation the nerve to the flexor carpi ulnaris should not be injured. One of us has made measurements of the amount that can be gained by changing the position of neighboring joints, but this is not the place for a detailed consideration of the subject.

6. *Technical Principles of Nerve Suture.*—We shall not in this paper enter on the finer details of nerve suture, but shall only mention some facts that are essential to this delicate surgical procedure. We believe that for nerve suture the best material is very fine silk (sepa-



Fig. 13.—Shrapnel injury of the posterior interosseous nerve. The end bulbs exposed.

rated strands of the finest silk obtainable) threaded on fine "Carrel" needles. For the actual union, perineurial and epineurial stitches are used. Epineurial sutures alone are not advisable. The funiculi are apt to retract—especially if a little blood or fluid collects between their

ends—and the resulting scar tissue will offer an impediment to the downgrowth of the regenerating axis cylinders from the central end (see Figure 9). After the nerve pattern of each end of the divided nerve has been carefully noted, one, two or three fine stitches are passed through the perineurial tissue—the number depending on the

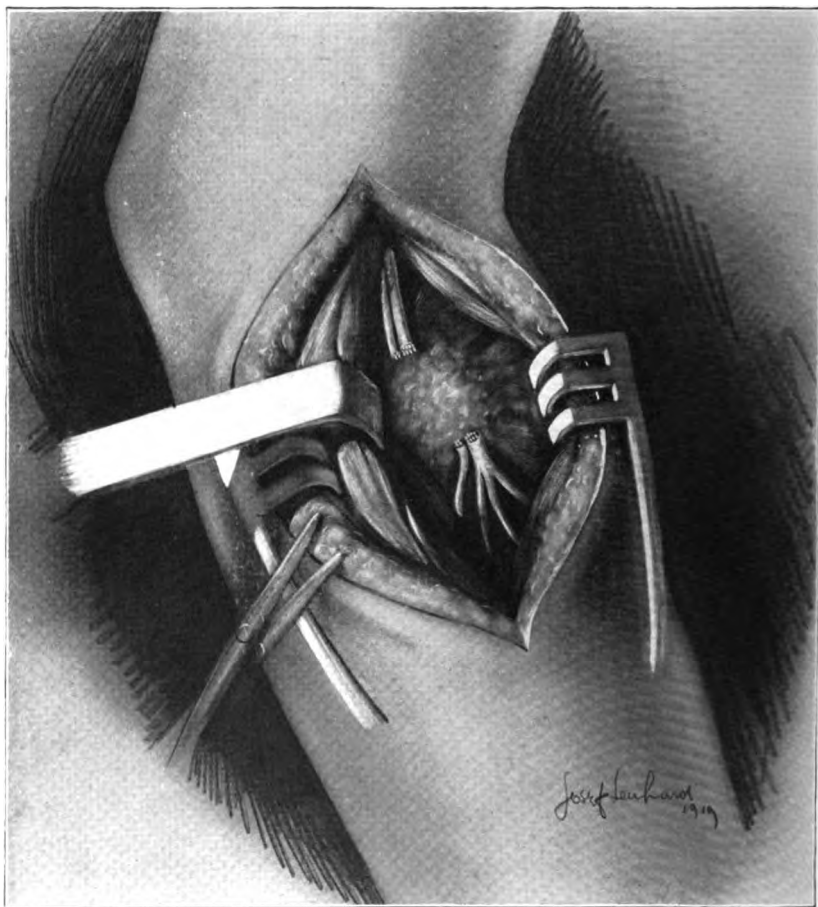


Fig. 14.—Same case as Figure 13. The end bulbs resected, showing the funicular pattern of the ends of the nerves.

arrangement and number of funiculi. No especial difficulty is encountered in passing the stitches through the perineurial tissue between the funiculi or groups of funiculi if the needle and suture material be sufficiently fine. If the sutures are well placed with due regard to the nerve pattern, a fairly accurate apposition of the ends of the funiculi is possible. All of the perineurial sutures should be passed before any

one of them is tied. When the sutures are tied, care should be taken that the funiculi are just brought into apposition. If the sutures are too tightly drawn, the funiculi are bent at their ends with a resulting poor approximation. After the perineurial sutures have been tied, the epineurial stitches must be accurately passed so as to approximate the free borders of the epineurial sheath.

Nerve suture, according to the principles we have outlined, can be done not only on the large nerve trunks, but also on smaller nerves. Thus one of us has been able to recognize the funicular arrangement

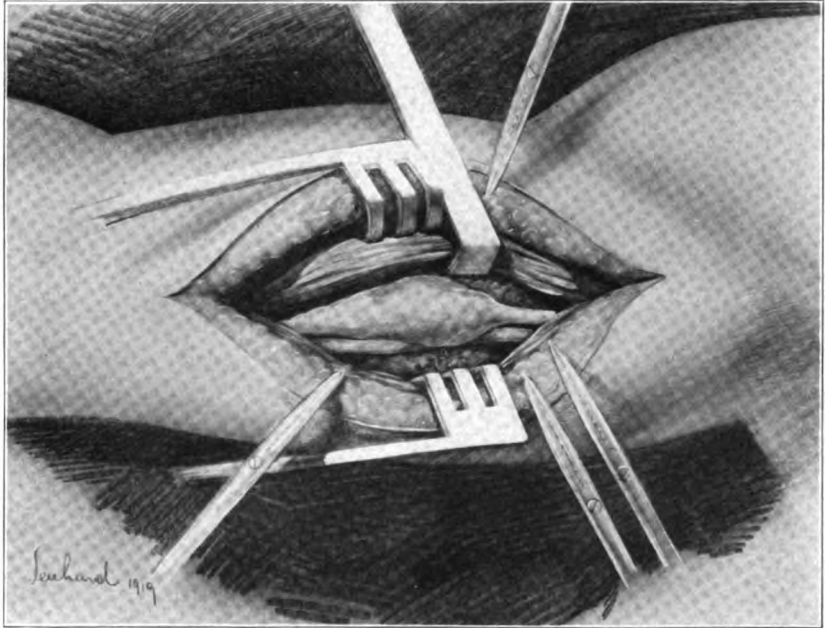


Fig. 15.—Large neuroma following shrapnel wound of median nerve.

and do a perineurial and epineurial suture on the internal cutaneous and lesser internal cutaneous nerves, on the deep palmar branch of the ulnar and on a large muscular branch to the triceps muscle from the musculospiral nerve (see Figure 10, 11, 12, 13, 14).

7. Concerning Procedures When Nerve Suture Cannot Be Done Because the Ends Cannot Be Approximated; Nerve Grafting.—If the condition of a nerve permits it, a neurolysis is always better than a resection and suture, and a resection and end to end suture far better than a resection and grafting.

If by changing the position of neighboring joints or by even moderate tension the ends of the resected nerve could be brought directly together, that was done in our patients in preference to introducing grafts; for the graft is for man as yet an unproved expedient. When the divided ends could not be brought together by reasonable tension and the other expedient elsewhere mentioned, grafts were employed, because nothing else was possible. In such cases the effort was made, whenever feasible, to replace each fasciculus of the main nerve with a fasciculus of the graft (see Figures 15, 16, 17).

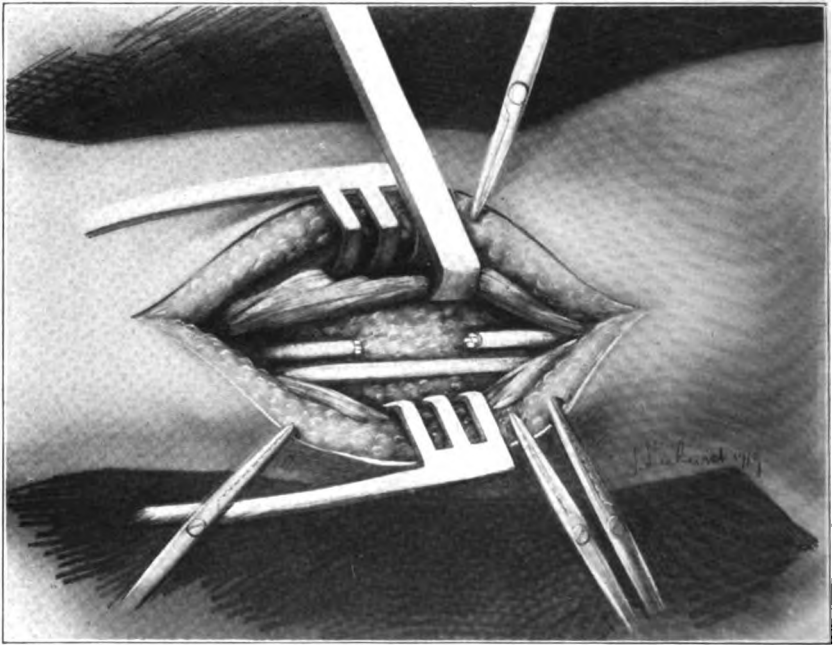


Fig. 16.—Same case as Figure 15. The neuroma has been resected. A defect 5 cm. long remained after the ends of the nerves had been brought as near as possible to each other. Note the funicular pattern.

We shall not enter into a discussion of the comparative value of autografts, homografts and heterografts, and of homografts preserved in cold storage, or in alcohol (Nageotte). Animal experiments have demonstrated that the autograft is to be preferred, and we have limited ourselves to the use of autografts of cutaneous nerves (external cutaneous of thigh; internal saphenous; anterior and posterior cutaneous branches of musculocutaneous; internal cutaneous). These grafts

III. THE OUTLOOK FOR THE FUTURE

In this paper we have considered only a few aspects of the symptomatology and of the anatomy and surgery of traumatic lesions of the peripheral nerves. The experiences of the world war will soon give us extensive statistics of the results that can be attained. It will be a difficult matter, however, to correctly judge of the end results when they are reported, for the technic of the operation has varied a great deal in the hands of different operators. It will require very careful discriminative studies of the end results when they are reported to determine to what degree the surgical methods used will have influenced the results obtained. We believe that in the advances that have been made, an important part is due to a fuller realization of the need for a better knowledge of the minute anatomy of the peripheral nerves. There can be no question that, no matter how much nature will help the surgeon, the surgical treatment of injuries to the peripheral nerves must be founded on the effort to bring the ends of a divided nerve into an apposition which approximates as much as possible the relation that existed before the injury occurred. With this aim in view, a very thorough knowledge of the structure of the peripheral nerves, of the course of fibers in the different funiculi, of the number and arrangement of the funiculi and of the functions of the fibers in each bundle is essential. Much attention has been paid to the course of fibers and fiber tracts in the brain and spinal cord, but a knowledge of the course of fibers in the peripheral nerves must still be acquired. The suggestions made in this paper constitute an effort in that direction. We believe that real peripheral nerve surgery will date from such a knowledge, and that the peripheral nerve surgery of the future, based on a minute and detailed knowledge of the finer anatomy and physiology of the nerves and a consequent advance in the finesse and delicacy of technic, will be able to show results far better and more complete than those that have been attained in the past. "*Natura non fecit saltum.*"

OVERLAP OF SO-CALLED PROTOPATHIC SENSIBILITY AS SEEN IN PERIPHERAL NERVE LESIONS *

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INTRODUCTION

For many years it has been noted that total loss of sensation after complete division of a peripheral nerve is limited to a much smaller area than we would expect from its anatomic distribution. Likewise, it has been observed that following injury of a peripheral nerve sensory symptoms may rapidly diminish and at times loss of sensation to pin prick be entirely absent.

That severe widespread anesthesia results only from trauma of several nerve trunks of a plexus, has generally been accepted. Lesions of single nerves result in partial anesthesia or, if a severe anesthesia be present, the area of complete loss of sensation rapidly shrinks.

Many attempts have been made to explain these phenomena. Among the older theories were: (1) That nerve fibers grow from healthy surroundings into the insensitive parts;¹ (2) that after section of a nerve stimulation of the severed part may pass through an accessory branch into an adjacent nerve and reach the major branch of the injured nerve above the lesion, through a second lateral branch (collateral innervation);² (3) that numerous anastomoses connect the peripheral ramifications of sensory nerves, many cutaneous areas receiving their innervation from different nerves. All these opinions have undergone important changes since the investigations of Head and his co-workers. The results of their brilliant studies led Head, Rivers and Sherren³ to conclude that "the sensory mechanism in the peripheral nerves consists of three systems:

1. "Deep sensibility, capable of answering to pressure and to movement of parts and even capable of producing pain under the influence of excessive pressure, or when the joint is injured. The fibers, subserving this form of sensation, run mainly with the motor nerves, and are not destroyed by division of all the sensory nerves to the skin.

* From the Department of Neuro-Surgery, U. S. Army General Hospital No. 28, Fort Sheridan, Ill. Under the direction of Lieut.-Col. Dean D. Lewis.

1. Schuh: Quoted by Oppenheim, Textbook on Nervous Diseases, Chicago, Chicago Medical Book Co. 1:408, 1911.

2. Letievant: *Traite des sections nerveuses*, Paris, 1873.

3. Head, Rivers and Sherren: *Brain* 28:111, 1905.

2. "Protopathic sensibility, capable of responding to painful cutaneous stimuli, and to extremes of heat and cold. This is the great reflex system, producing a rapid, widely diffused response, unaccompanied by any definite appreciation of the locality of the spot stimulated.

3. "Epicritic sensibility, by which we gain the power of cutaneous localization, of discrimination of two points, and of the finer grades of temperature, called cool and warm."

Head and Sherren⁴ state that in complete division of a mixed nerve, as the median or ulnar, the area it supplied does not become uniformly insensitive. Whereas previous observers have stated that sensation is diminished over the full area usually assigned to the injured nerve and lost completely over a small portion only, they have shown that this diminution of sensation is in reality a total loss of sensibility to stimulation with cotton wool, to the compass test, to the painless interrupted current, and to degrees of temperatures between 22 C. and 40 C. In this area are felt only the stimuli affecting the protopathic sensibility, such as the prick of a pin and temperatures below 20 C. and above 40 C. The area rendered insensitive to light touch by division of the median or of the ulnar nerve varies little in extent. In sharp contrast to this slight variation is the extreme difference in surface extent of the loss of sensation to a pin prick which follows division of either of these nerves. "The consequence of both division and irritation of these nerves shows that as far as protopathic sensibility is concerned they overlap to an enormous extent."⁵

It is evident, therefore, that the complete sensory distribution of a peripheral nerve consists of its exclusive supply, or that area in which loss of sensation is produced by its division, and in addition its overlap or the area determined by the limits of skin sensitive to stimuli when all the adjacent nerves have been severed. Head and Sherren, employing the method of residual sensibility, were able to determine the complete sensory distribution of some of the nerves. These areas were part of the median, the internal saphenous, part of the external popliteal, the external saphenous and part of the posterior tibial.

The purpose of this paper is: first, to record the smallest area of loss to prick pain which follows interruption of the various peripheral nerves; second, to point out the relative smallness of this area as compared to the area of loss to touch; third, to show that the preservation, or early return of prick pain as compared to tactile sense is due to the assumption of function of adjacent nerves and not to nerve regeneration, as interpreted by Head; and, fourth, to outline the total sensory distribution of some of the peripheral nerves by residual sensibility.

4. Head and Sherren: *Brain* **28**:283, 1905.

5. Head and Sherren: *Brain* **28**:295, 1905.

MATERIAL

My observations were made on 500 patients with peripheral nerve lesions seen early in base hospitals in France, and 520 patients with peripheral nerve lesions studied later at U. S. Army General Hospital No. 28, Fort Sheridan, Ill.

The observations of early peripheral nerve lesions were in most instances uncontrolled by operative procedures. A large proportion of the lesions were partial and frequently complicated by injuries to adjacent small sensory branches. But these observations served a useful purpose. They showed: (1) that in many cases for the first two or three weeks only a very small area within the border of the part insensitive to cotton wool was sensitive to pin prick; (2) that in a few a larger zone sensitive to pin prick appeared within fifteen days; and (3) that the return of sensitiveness to pin prick in a larger zone, corresponding to the area which we later determined as overlap, usually was found, at times variable from thirty to one hundred days. The cases showing return to pin prick over a large area in less than thirty days were predominantly cases of radial and musculospiral lesions.

The material of peripheral nerve lesions studied later may be divided into two groups: the first, a group of 391 cases of peripheral nerve lesions uncontrolled by operation, and in the majority of instances recovering spontaneously; the second, a group of 129 cases controlled by operation.

CASES CONTROLLED BY OPERATION

Nerve Lesions	Number
Ulnar	20
Radial	31
Median	9
Ulnar and median.....	15
Median and radial.....	2
Musculocutaneous, ulnar and median.....	2
Brachial plexus	2
Great sciatic	20
External popliteal	25
Anterior tibial	2
Ulnar, median and internal cutaneous.....	1
Total	129

My general impressions relative to the sensory changes in peripheral nerve lesions were derived from the whole material. The areas of total nerve supply and of overlap were obtained only from cases certified by operation. The cases which have been used in the study of regeneration of nerves likewise were certified by operation.

Therefore, although the whole 1,020 cases contributed to my general conclusions concerning these problems, only one group, consisting of the cases coming to operation, was employed in obtaining the data which serve as the basis for the special conclusions contained in this article.

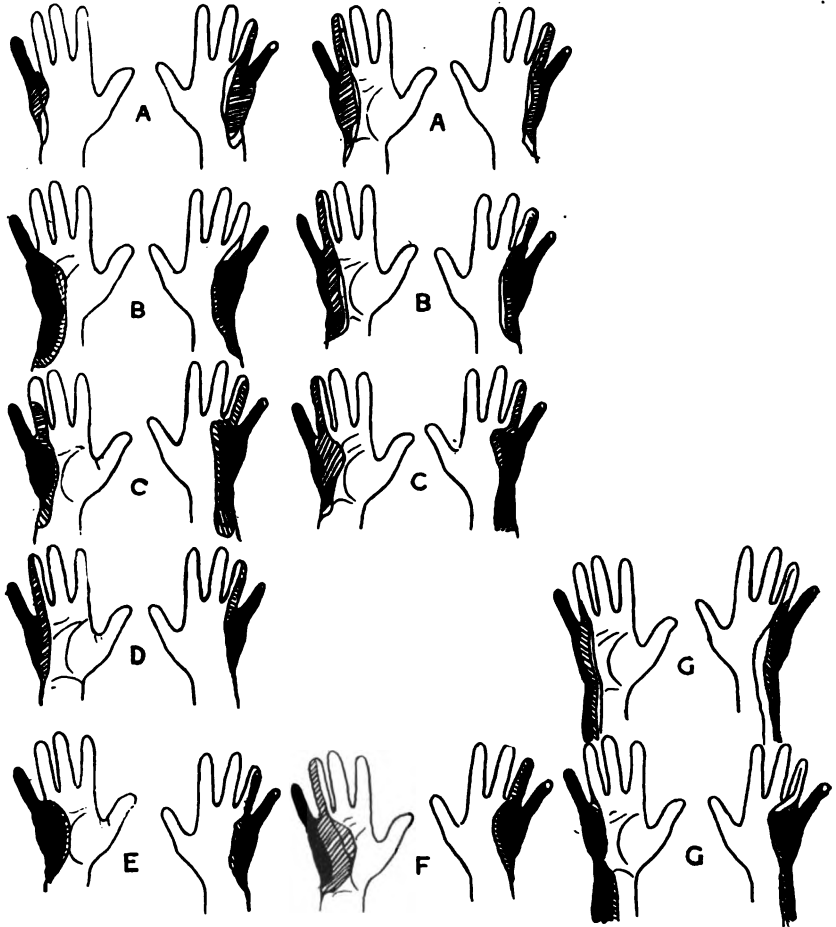


Fig. 1.—Sensory changes in ulnar nerve lesions: Diagonal lines, anesthetic to touch; black lines, loss of prick pain and touch sense; continuous line, borders of loss of temperature sense. The same scheme of charting is followed in all of the diagrams. Where duplication of letters occurs the first is preoperative and the second postoperative sensory chart.

METHODS OF INVESTIGATION

The problems under investigation were studied from a clinical and not from a psychophysical standpoint. The areas of overlap were found in the course of clinical examinations of a large group of cases. The methods of examination, therefore, were those ordinarily used

clinically. The sense of touch was tested by a wisp of cotton. The sensation of pain in response to the prick of a pin was ascertained by using a weighed needle sliding within a bit of glass tubing, so that with different weighed needles a pressure of from 5 to 35 gm. could be applied.

Although in this report temperature sense will not be referred to because of difficulty of standardizing methods and the impossibility of employing the finer methods clinically, it may be stated that for the rough examination of sense of cold, we used a pledget of cotton twisted to a point and saturated with ether. Ether has been employed in the form of droplets by von Frey. We have found that our method per-

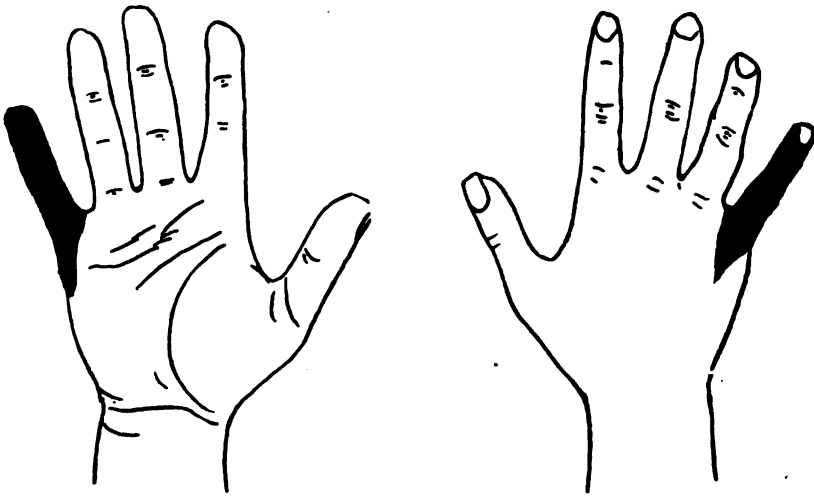


Fig. 2.—Smallest composite area of analgesia in ulnar nerve lesions.

mits a less diffuse type of stimulation and has the advantage of ease and simplicity. For physiologic research this method is, of course, inapplicable and therefore our results will not be incorporated in this discussion.

It must be determined whether our methods are acceptable as to the differentiation of so-called epicritic and protopathic senses and deep sensibility. Light touch with a wisp of cotton to determine sense of touch may, I think, be accepted if exact threshold of sensation is not under investigation, and if exact borders of loss of sense of touch be not insisted on. For the purposes of this investigation, the exact borders of loss of sense of touch need not be insisted on. Only one factor must be considered in this method of examination, namely, return of so-called hair sensibility must not be confused with touch; hence, in testing for touch where an accurate border was to be determined, the parts were closely shaven.

The degree of pressure which it is permissible to employ in determining prick pain without jeopardizing the results by confusion with pressure pain remains to be discussed. Although, as pointed out by Head and Sherren,⁶ deep sensibility may be evoked when testing for touch with a stiff roll of wool, this objection is not valid for determining prick pain, within certain limits. A sharp needle was used by Head and Sherren in their early clinical investigations, care being taken to

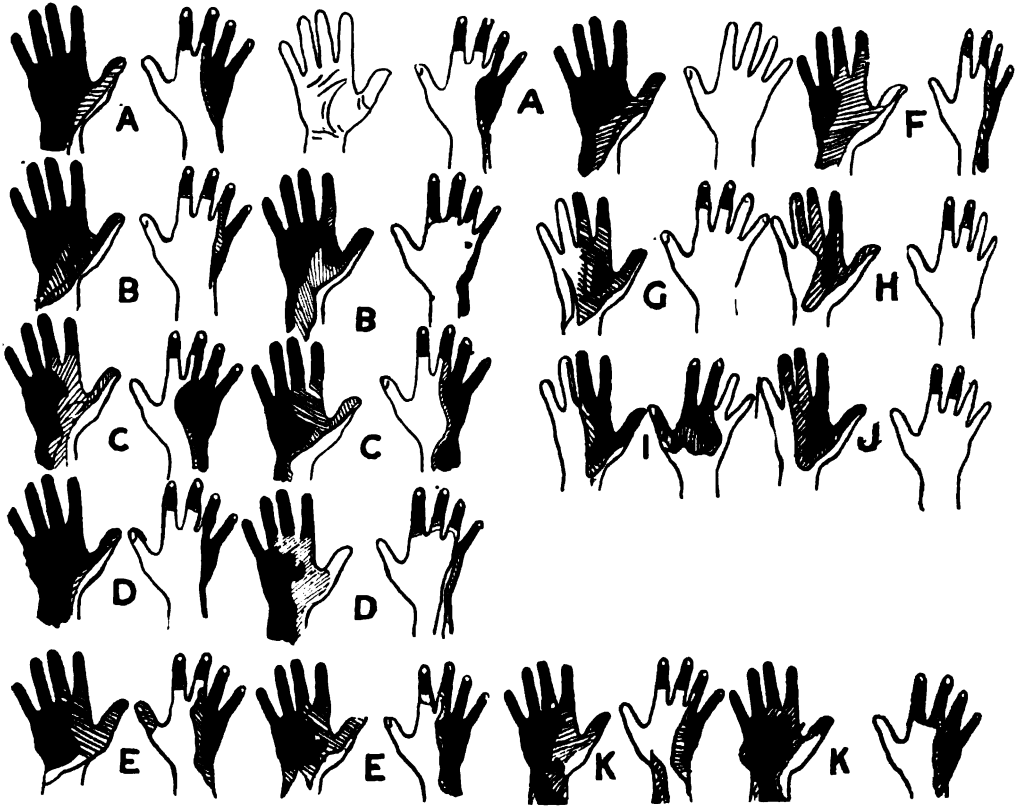


Fig. 3.—Sensory changes in median nerve lesions: *g, h, j*, radial and median; *i*, ulnar and median; *b, e*, ulnar, median and internal cutaneous; *a, c, d, e*, ulnar, median, and musculocutaneous, *k*.

differentiate between sense of deep pressure and true pain. Boring⁷ says that "In determining the pain threshold it was especially necessary not to exceed pressures of 5 gm. Although at high intensities of stimulus the introspective difficulty of abstracting from pressure was less with pain than with cutaneous pressure, the greater intensities

6. Head and Sherren: *Brain* **28**:120, 1905.

7. Boring: *Quart. J. Exper. Physiol.* **10**:16, 1916.

frequently drew blood and therefore were abandoned." As in Boring's work it was necessary to examine a small area of skin repeatedly and at very short intervals for all forms of sensation, his objection is valid. In my case, on the other hand, it was necessary only to examine sense of prick pain in areas of overlap and not to confuse this pain with pressure pain. We have never found pain to result from 35 gm. of pressure with a blunt object, and care being taken to obtain from the patient responses only to pain from prick of a sharp point, I believe pressure of even 35 gm. to be permissible to map out the overlap of sense of prick pain. No exact measurements of threshold to prick pain were made and in the majority of cases pressure did not exceed 30 gm.

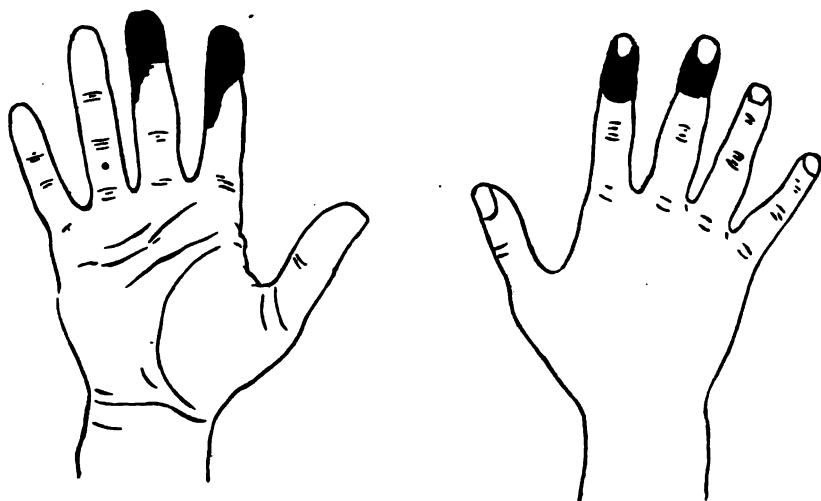


Fig. 4.—Smallest composite area of analgesia in median nerve lesions.

1. EXCLUSIVE NERVE SUPPLY

As I recognized that following section of a mixed nerve the loss to prick pain occupies an area much smaller than the loss to touch, the first part of my task was to ascertain the smallest area which is insensitive to pin prick following section of various nerves. This would indicate the limits of any possible overlap.

Although only a small portion of the area insensitive to touch is quite insensitive to pin prick, diminution of pain sense is present in a large part of the area insensitive to touch, and if graduated degrees of pressure be employed, concentric rings of analgesia are demonstrated. However, we are concerned not with the question of whether any hypalgesia is present, but whether any portion of the skin in the recognized anatomic sensory distribution of a nerve is at all sensitive to pain, provided this pain be due to superficial sensibility. If a part of

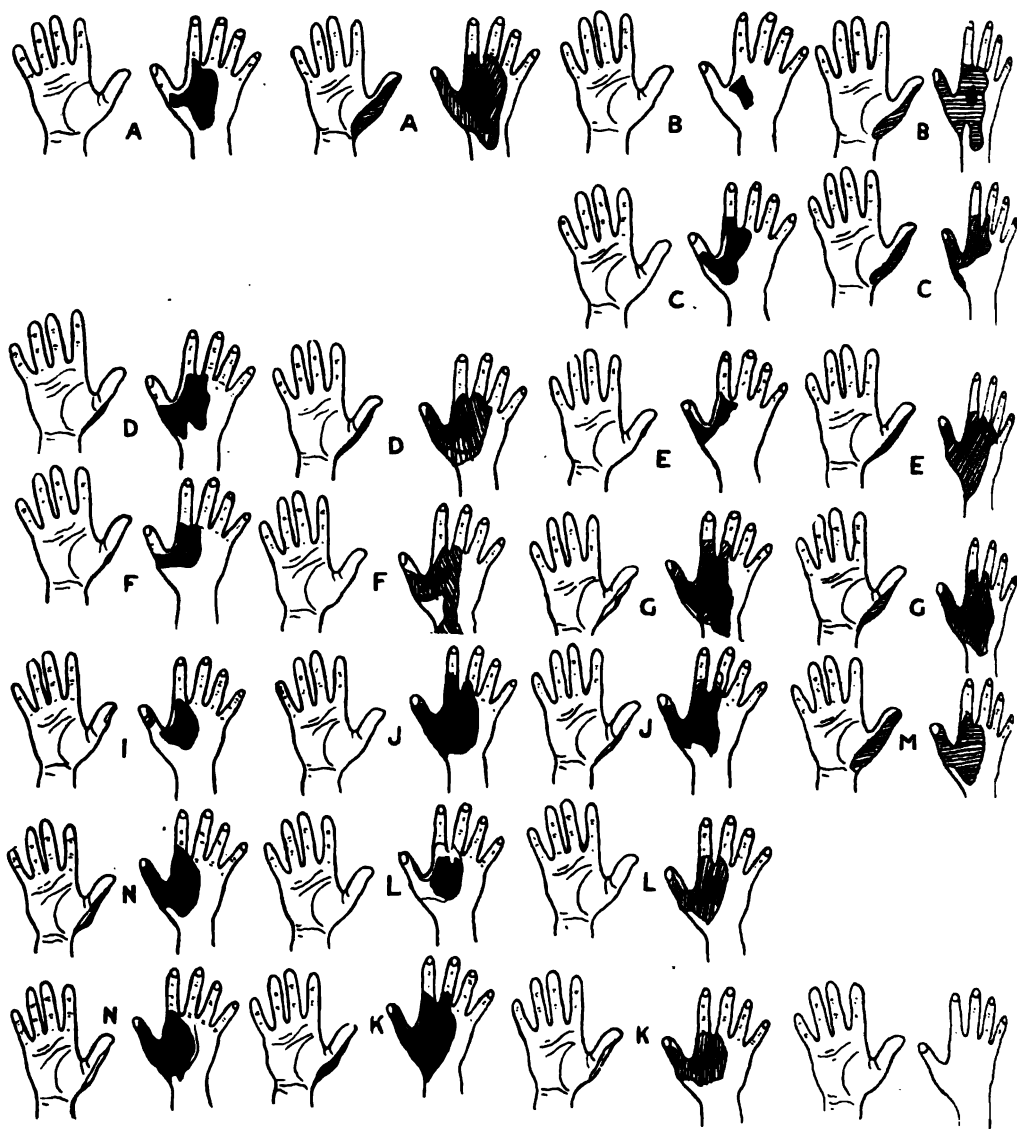


Fig. 5.—Sensory changes in radial nerve lesions.

the skin is sensitive to pain, when a nerve is divided, this sensation must be derived from some source other than this nerve.

To delineate the area exclusively supplied with pain sense by a given nerve one of two conditions must be present: First, the presence of pain sense having been demonstrated within the area of a nerve's supposed anatomic supply, that nerve is found at operation to be divided and the ends separated. Second, the nerve having been seen to be divided, presence of pain sense is demonstrated in its distribution

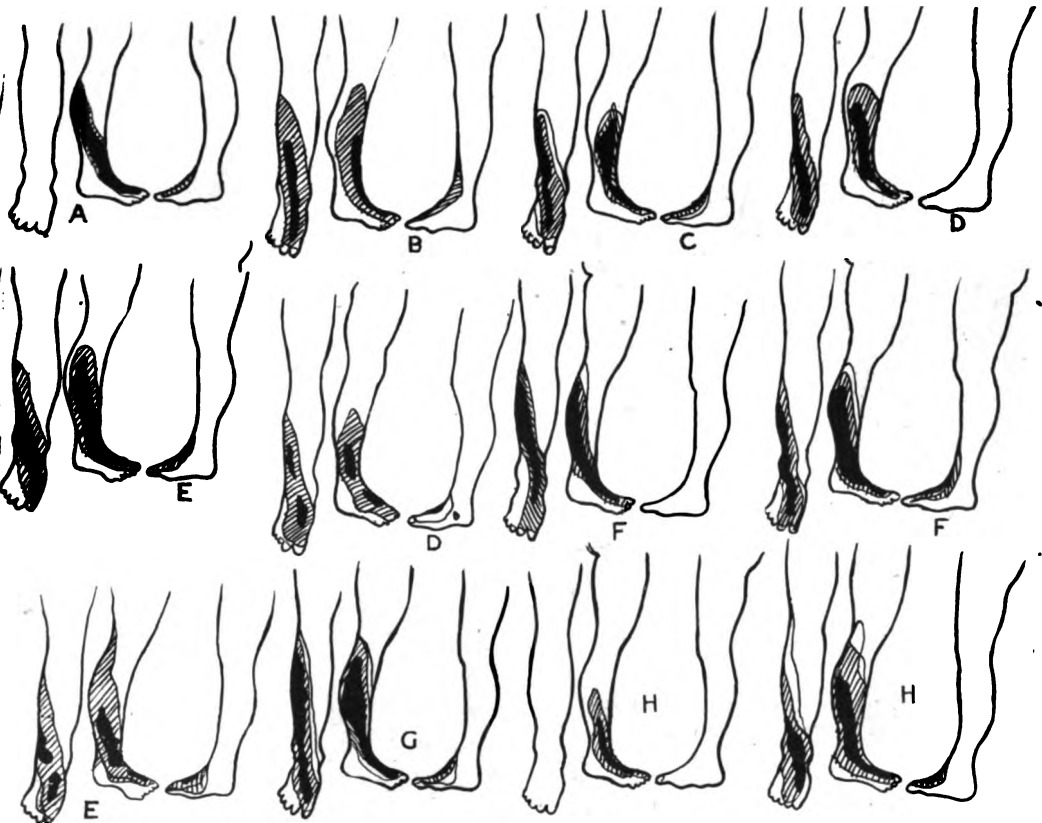


Fig. 6.—Sensory changes in external popliteal lesions.

within the length of time given for the return of protopathic sensibility (Head, Rivers and Sherren, forty-three days). In my cases, under the second condition twenty-eight days was the limit, with the exception of the radial nerve in which the limit was thirty-seven days.

The relatively few cases studied does not make it profitable to attempt to outline the exclusive supply of peripheral nerves to both epicritic and protopathic sensibilities. Suffice it to say that our results

as to the nerves in the hand are in general accord with Stopford,⁸ who found in the ulnar nerve some variation from the accepted area of epicritic sense in 20 per cent. of the cases and in the median nerve, in 38 per cent. In three of my cases of median nerve section anesthesia was present over the dorsal surface of the distal phalanx of the thumb.

In ulnar nerve lesions superimposing the outlines of complete analgesia, in the cases shown in Figure 1, *a, b, c, d, e, f* and *g*, the smallest area of analgesia was found to occupy the palmar and dorsal surfaces of the little finger, extending over the dorsal surface of the hand in a triangular area over the fifth metacarpal bone to one-third of its length (Fig. 2). The area included between the borders of the accepted sup-



Fig. 7.—Smallest composite area of analgesia in external popliteal lesions.

ply of the ulnar nerve and the borders of this analgesia represents the possible supply of overlapping nerves to pain sense.

The inner border of the smallest area of exclusive supply to pain of the median nerve was obtained in the same way from cases in which the median nerve was subsequently found to be divided (Figure 3, *g, h, i, j*). The outer border was obtained from these cases and in addition from cases of combined ulnar and median lesions which at operation were likewise found to be anatomic divisions, with the ends separated (Fig. 3, *a, b, c, d, e, f, k*). The exclusive supply of the median nerve to pain sense was found to occupy the dorsal and palmar surfaces of the distal phalanges of the index and middle fingers, the ulnar half of the palmar surface of the second phalanx of the index

8. Stopford: *J. Anat.* 53:14 (October) 1918.

finger, part of the ulnar portion of the distal half of the second phalanx of the middle finger and the dorsal surface of less than half of the second phalanges of the index and middle fingers. Despite the fact that this small area of total analgesia in median nerve lesions has been recognized,⁹ it is necessary at this point to call special attention to this observation as from the study of this nerve much of our evidence relative to overlap was obtained (Fig. 4).

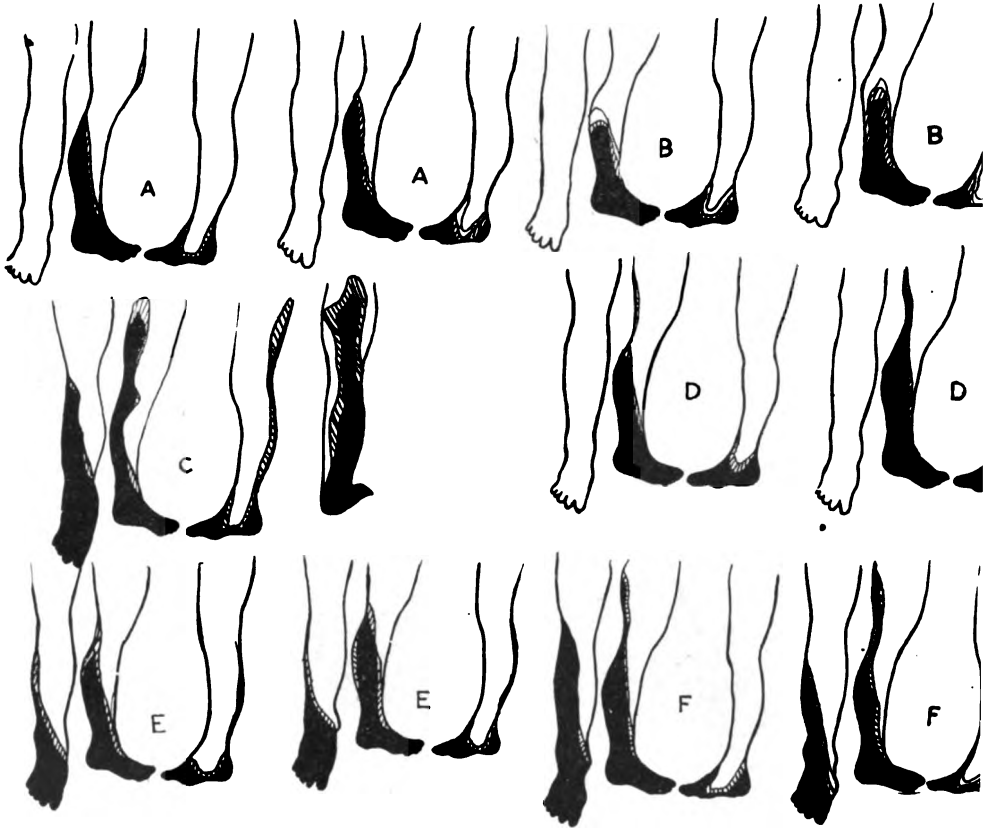


Fig. 8.—Sensory changes in sciatic nerve lesions.

The cases of radial nerve lesions, certified at operation or examined less than thirty-seven days after resection and suture, showed a wide variety of areas of analgesia and in one case no analgesia at all. (Fig. 5, *a* to *m*). Head and Sherren's case 44 of division of the radial nerve where it passes under the tendon of the supinator longus, likewise showed no loss to prick pain.

9. Benisty, Athanassio: *Clinical Forms of Nerve Lesions*, London, 1918; Head and Sherren: *Brain* 28:136, 1905.

Although not infrequently recorded, we have not observed any case of radial nerve lesion which did not show loss of sensation to touch. Of all the peripheral nerves, the radial shows the greatest variation in the areas of loss of sensation to both epicritic and protopathic sensation. This is due to the fact that six nerves are concerned with the sensory supply of the dorsum of the hand; the median, radial, anti-brachii posterior branch of the musculospiral, musculocutaneous and ulnar.



Fig. 9.—Smallest composite area of analgesia in sciatic nerve lesions.

Stopford⁸ emphasized, as did Head and Sherren, the importance of the musculocutaneous nerve in the supply of the dorsum of the hand, and states that its terminal branches may extend on to the dorsum of the metacarpus, and "it appears that the extent of its distribution varies inversely with that of the radial." Although this may be true, it must not be forgotten that the median nerve must be considered in the supply of sensation to the dorsal area over the distal portion of the metacarpus and the distal portion of the thumb.

One of the reasons for varying reports relative to the sensory loss in radial nerve lesions is the hairy nature of the area of skin under

investigation. The early return of hair sensibility frequently is confused with the presence of sense of touch. The skin must be closely shaven in all cases where examination of touch sense is contemplated. In our charts, where touch is outlined, this precaution was taken. Where examinations were made in the presence of hair, areas sensitive to "touch" frequently were analgesic.

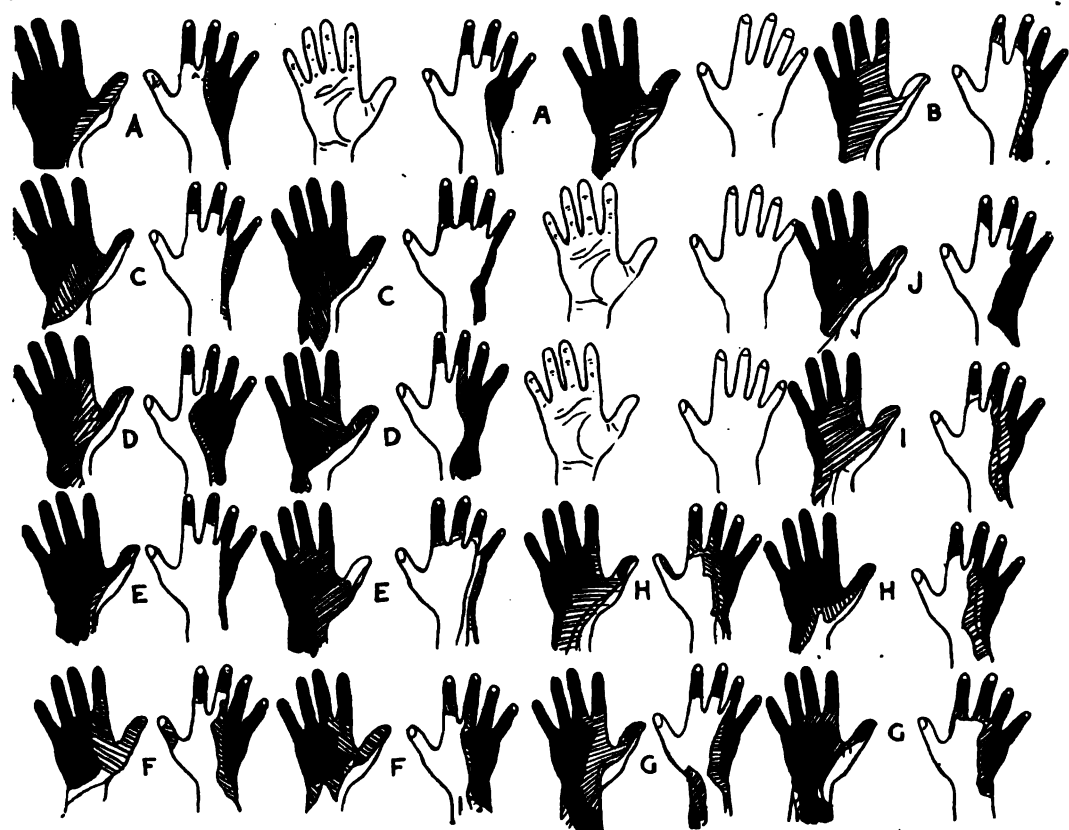


Fig. 10.—Sensory changes in combined lesions of the ulnar, median and internal cutaneous nerves.

No area of skin is exclusively supplied by the radial nerve for prick pain.

The area of exclusive supply of pain of the external popliteal nerve was obtained from certified cases of division and cases examined less than thirty-seven days following resection and suture (Fig. 6, *a* to *h*).

The area consists of a narrow band extending from a point a little above the junction of the lower and middle one-third of the outer surface of the leg, diagonally across the dorsum of the foot to a point

over the middle of the metatarsal bone of the great toe. It is interrupted at the junction of its lower and middle one-third by an area which is sensitive to pin prick. The area is due to the overlap on one side of the internal saphenous nerve and on the other side, the internal popliteal nerve. Although a number of cases showing such an interruption in the band of analgesia have been observed, they have not fulfilled the requirements which we demanded in estimating exclusive supply. One case, Figure 6, *e*, showed this type of interruption of the band of analgesia twenty-seven days after resection and

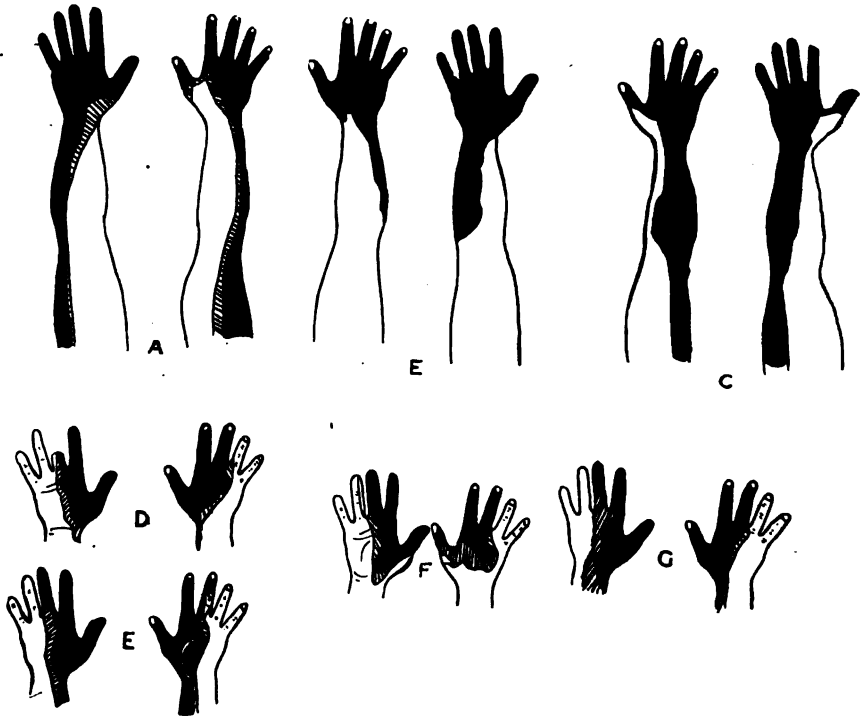


Fig. 11.—Sensory changes in combined lesions of the ulnar, radial and median nerves, *a*, *b*, *c*, and of the median and radial, *d*, *e*, *f*, *g*.

suture. Another case which was examined fifty-three days after resection and suture is shown in Figure 6, *d*, but was not used in estimating the isolated supply.

Our results, therefore, are at variance with Head, Rivers and Sherren,³ who, referring to the fact that the unit for protopathic sense is the posterior root, said that following section, the external popliteal, representing a large part of the fifth lumbar root, does not show a great difference between the borders of the areas of analgesia and anesthesia.

The external popliteal nerve has a surprisingly small exclusive area of pain sense (Fig. 7).

The area of the sciatic nerve was obtained from cases certified to be anatomic divisions, Figure 8, *a* to *f*. This area is illustrated in Figure 9 and need not be described.

Inasmuch as the results above illustrated represent the smallest area of exclusive supply of various nerves for pain, it is necessary to define to what extent they may be used in formulating our ideas

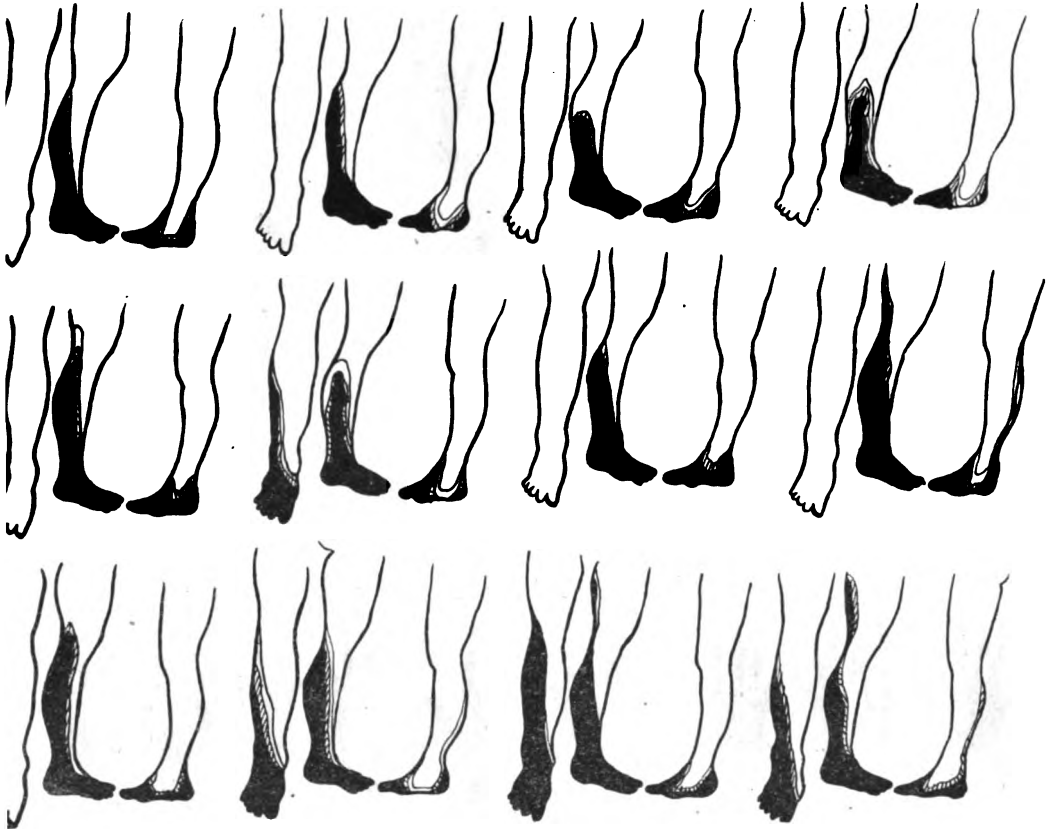


Fig. 12.—Sensory changes in combined lesions of internal and external popliteal portions of sciatic nerve.

relative to nerve overlap. I recognize that in some instances such small areas may be present only when we are dealing with the group of 25 per cent. of cases showing unusual distribution of sensory nerves. These areas are used, therefore, only in establishing a certain limit beyond which it is not permitted to go in interpreting return of sensation to pain as a sign of nerve regeneration. Any return of sense of pain in regions without these borders may be due to unusual nerve

distribution or sensory overlap, and represents possible areas of overlap. It will be found that the areas of overlap I will later describe are not as extensive as these areas would permit us to assume were we to use exclusive pain sensibility as an indication of the borders of overlap.

2. NERVE OVERLAP

I maintain that the return of sensibility to pin prick, which occurs before the return of sensibility to touch, occurs in regions which

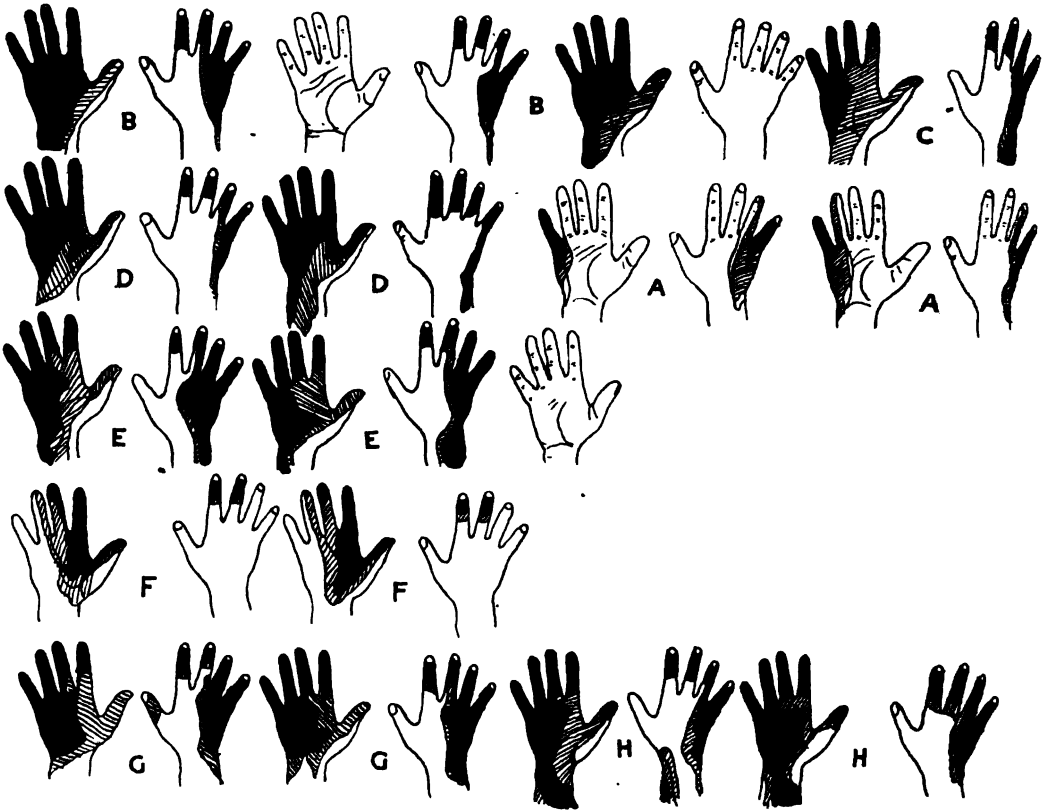


Fig. 13.—Sensory changes before and after resection and suture of the ulnar, median and ulnar and median nerves.

occupy the areas of nerve overlap, and that this return of sensibility to pin prick cannot be interpreted as a sign of nerve regeneration.

I am supported in this view by the facts that I have never found a return of sensibility to pain, when sensibility to touch has not returned, except in an area of overlap: that when a nerve is divided and at the same time one or more adjacent nerves are divided sensation to pin prick does not return in the area of the overlap of these

nerves even many months following the injury; that when a nerve adjacent to one which is severed and which supplies an area of overlap to that nerve is sectioned, the preexisting sensibility to pin prick in the overlap area is lost; that when sensibility to pin prick is present within the anatomic sensory distribution of a severed nerve resection and suture has no effect on the general outline of this area of sensibility.

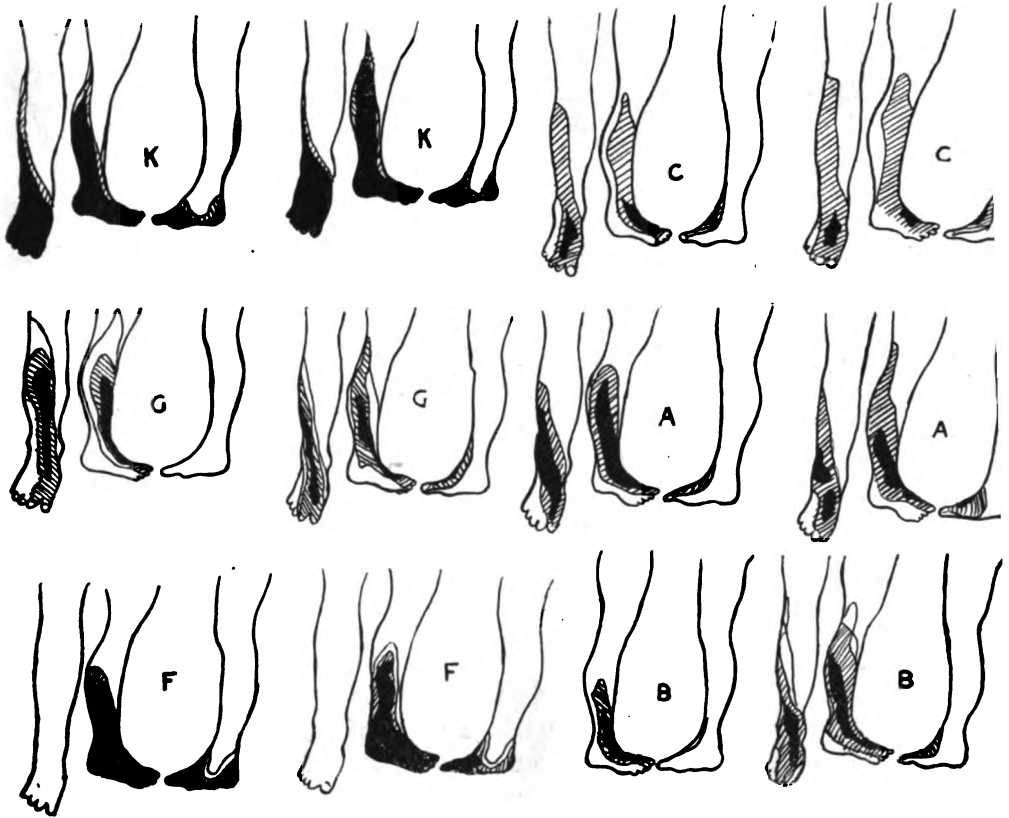


Fig. 14.—Sensory changes before and after resection of external popliteal and sciatic nerves.

The statement of Head, Rivers and Sherren¹¹ that “the length of the nerve to be regenerated makes relatively little difference to the time at which protopathic sensibility returns,” only strengthens my thesis, for were the return of protopathic sensibility due to regeneration of protopathic fibers, this process of regeneration would have to conform with the same temporal law of regeneration as do the fibers carrying epicritic impulses, however rapidly the protopathic fibers might regenerate.

10. Head, Rivers and Sherren: *Brain* 28:108, 1905.

Within two weeks after the occurrence of a peripheral nerve lesion the area of analgesia usually nearly coincides with the area of anesthesia. Some cases showed an intermediate zone or a shrinkage of the analgesic area within fifteen days. In from thirty to one hundred days the majority of cases showed the presence of a shrinkage to an extent which was later identified with overlap. It is probable that my cases would have shown the same extent of shrinkage in less than a hundred days, but conditions were such that in these cases the first record available was obtained one hundred days after the injury. Certainly the majority of cases showed the shrinkage to be well established under fifty days. Some months after the injury had been received the shrinkage was present and the remaining area of analgesia has been described above as the exclusive sensory supply for pain sense in various peripheral nerves.

The shrinkage of the analgesic area can be due to but two conditions — nerve regeneration or assumption of function by adjacent nerves.

If any overlapping of peripheral nerves is possible, it becomes necessary to define the extent of this overlap before any return of sensation can be interpreted as a sign of nerve regeneration.

Head, Rivers and Sherren¹¹ noted the presence of overlap of so-called protopathic sense, as the following quotation indicates: "Enormous overlapping occurs as we have already seen from a consideration of the analgesia caused by division of the median or of the ulnar nerves." Despite their recognition of overlap, they make no attempt to separate it from return of sensation due to nerve regeneration. Apparently they group both together when they say (page 108): "If the nerve has been completely divided, protopathic sensibility returns first, followed at a considerable later period by return of epicritic sensation." Speaking in this instance of recovery of sensation, they then go on to say without any reservation that: "Evidently, the two systems regenerate with unequal facility. The protopathic system regenerates more rapidly and with greater ease." Nor do Head and Sherren¹² clearly differentiate the two processes. Speaking of recovery of sensation after division of the nerves of the hand, they say, "But the ill defined borders and the comparatively small extent of total analgesia, and the fact that a large part of the palm rarely becomes insensitive to prick from a lesion of one nerve only, all point to much overlapping of the fibers that conduct pain impressions. Such overlapping should lead to rapid restoration of sensibility

11. Head, Rivers and Sherren: *Brain* 28:110, 1905.

12. Head and Sherren: *Brain* 28:148, 1905.

to prick, and in some cases possibly forms a factor when sensation returns with unusual rapidity. Commonly no wide loss to prick on the palm follows division of the median nerve, because the fibers which conduct this form of sensation are supplied from both nerves. But supposing the nerve supply of the median palm came overwhelmingly from the median, division of the nerve would produce at first total analgesia. This might rapidly pass away, to some extent, as soon

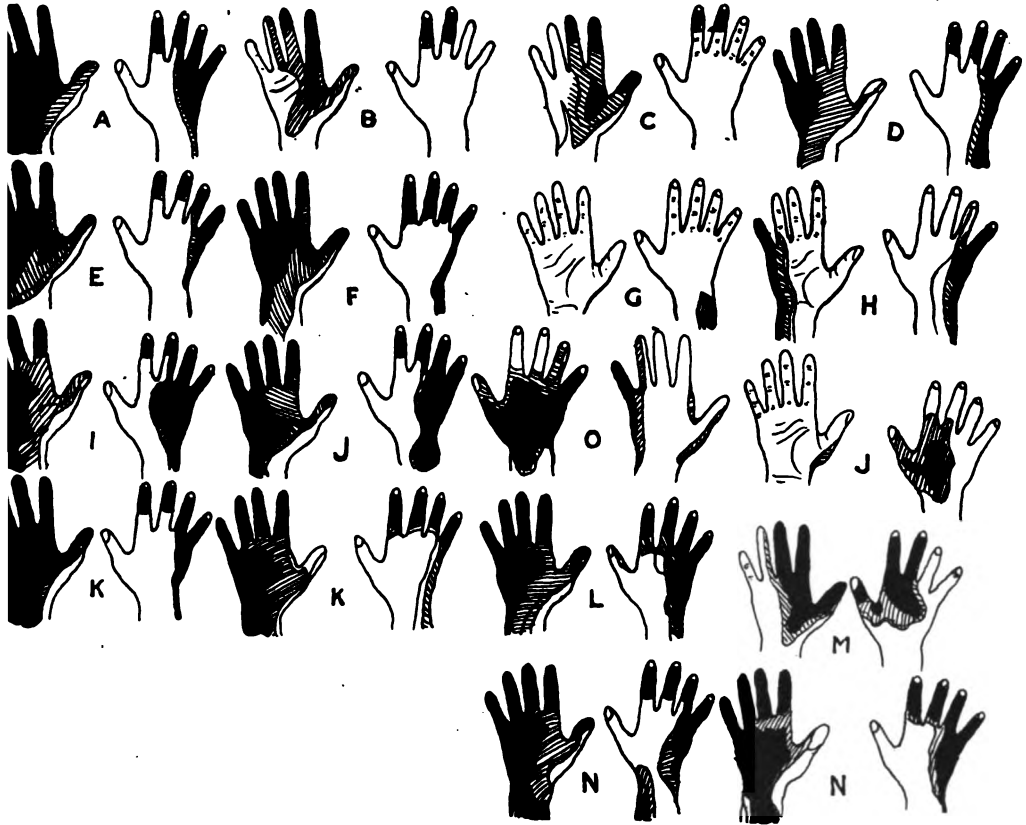


Fig. 15.—Sensory changes in lesions of median, internal cutaneous, combined median and radial nerve, *b, g, m*, from which the residual sensibility of the ulnar nerve was obtained; and of the ulnar and internal cutaneous, radial, combined radial and median nerves, *h, j, m*, from which the residual sensibility of the median nerve was obtained.

as the few fibers of the ulnar nerve to the median palm became capable of supplying sufficient sensibility for the transmission of impulses." Again they say (page 149): "With so much overlapping of nerve supply, complete recovery of sensibility to prick pain might occur without union of the divided nerve, by a further development of those

fibers in the uninjured nerve which normally supply the affected parts. In areas where sensation to prick is only partially lost, such substitution undoubtedly occurs, . . . But there is no evidence to show that restoration of sensation can be produced in analgesic parts without union of a divided nerve." Finally, they state (page 140) that, "Division of the nerve (one of the nerves of the hand) leads at once to the production of an area of absolute cutaneous insensibility, surrounded by an area of loss of sensation to stimuli, such as light touch and the minor degrees of temperature. The relative extent of these two areas differs greatly in each individual case, and the first definite sign of recovery is shown by an increase in size of the intermediate zone between them." So far as I can ascertain, no evidence has ever been adduced to show that overlapping nerves functionate immediately

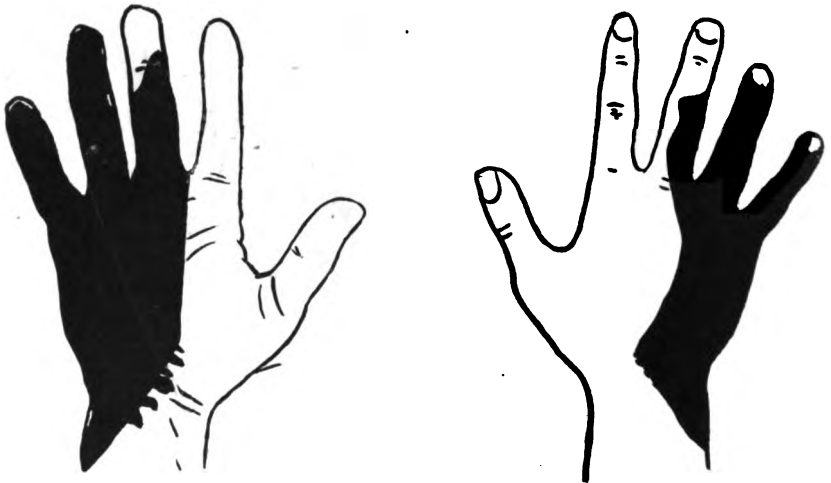


Fig. 16.—Residual sensibility to prick pain of the ulnar nerve.

following the injury of an adjacent nerve. Neither have the laws of dual innervation been clearly defined. Until this is accomplished, it is illogical to infer that return of sensation in the area of an overlapping nerve is a sign of nerve regeneration and is not caused by the functioning of this overlapping nerve.

If the shrinkage of the area insensitive to pin prick, responsible for the increase in size of the intermediate zone, be a sign of nerve regeneration and not a result of overlap, it should occur whether the adjacent nerves be intact or not. This, however, is not the case, as will be shown. In other words, if certain areas of skin become sensitive to pain or are found sensitive to pain following section of a given nerve, and the condition is due to nerve regeneration, then section of the adjacent nerve would have no effect on the appearance of this sensibility. What are the facts?

SECTION OF NERVES

Section of Adjacent Nerves.—Although in isolated lesions of the ulnar nerve sensibility to pain is frequently seen on the ulnar half of the ring finger, this is never observed when the median nerve is divided at the same time (Fig. 10, *a, f*).

Although isolated lesions of the ulnar and of the internal cutaneous nerves always show that the distal end of the analgesia resulting from a lesion of the internal cutaneous and the proximal end of the analgesia resulting from a lesion of the ulnar, do not meet, no instance is found in combined lesions of the ulnar, median and internal cutaneous nerves where an area between the borders of the analgesia of the internal cutaneous and ulnar nerves is sensitive to pain (Fig. 10, *b, c, d, e, h, i, j*).



Fig. 17.—Residual sensibility to prick pain of the median nerve.

When the ulnar, radial and median nerves are divided, a year may follow their division and no shrinkage of analgesia be found on the palmar or dorsal surface of the hand except on the proximal portion of the analgesia where the musculocutaneous and the antibrachii posterior areas adjoin the analgesic area (Fig. 11, *a, b, c*). When a radial lesion is combined with a median, analgesia is always present on the radial part of the palm. When a median lesion or a radial lesion alone is present, this part of the palm is usually sensitive to pin prick (Fig. 11, *d, e, f, g*).

Contrary to Head, Rivers and Sherren, who state that destruction of the external popliteal nerve (which corresponds closely to the fifth lumbar root) produces a widespread loss of sensation to prick, I have

found that isolated lesions of this nerve may show only a small area of analgesia, but when the internal popliteal as well as the external popliteal is severed, there is never found any shrinkage of analgesia or reappearance of sensibility to prick pain in the zone where the supply of the external popliteal meets that of the internal popliteal (Fig. 12, *a* to *g*).

I think it can be definitely stated that when nerves supplying adjoining areas are severed, sensation to pain is at no time present in the border areas where it is uniformly observed when either nerve is divided alone.

Inasmuch as a large number of my cases have had resections and sutures performed at least three months prior to the last examination,



Fig. 18.—Residual sensibility to prick pain of the musculospiral nerve.

it may be stated likewise that no sensation to pain returns in such areas in the time given for the beginning of regeneration of protopathic sensibility.

Effect of Section of an Overlapping Nerve.—When return to sensibility to pain or presence of sensibility to pain is found in the area of overlap of an adjacent nerve, analgesia will result if this nerve is severed.

This is well illustrated in the case shown in Figure 3, *g*. This patient had a partial ulnar lesion combined with a complete section of the median. Prick pain was preserved in the radial portion of the palm and the index finger. When at operation the superficial radial

nerve was resected for use as a cable transplant, this part of the palm became analgesic (Fig. 11, *e*).

Effect of Resection and Suture on Existing Overlap.—Head and Sherren¹³ state that "Sometimes it is necessary to divide an injured nerve, after sensibility to prick has already begun to return to the hand, that more perfect union may be obtained. Wherever such an operation has been performed, the parts that had begun to recover sensibility became again insensitive to prick, a proof that the recovery must have been due to union, however imperfect, of the divided nerves." This contention they support by Case 11 in which the ends of the divided nerve were united by what appeared to be fibrous tissue. After the operation, the extent of the total loss of sensibility to prick

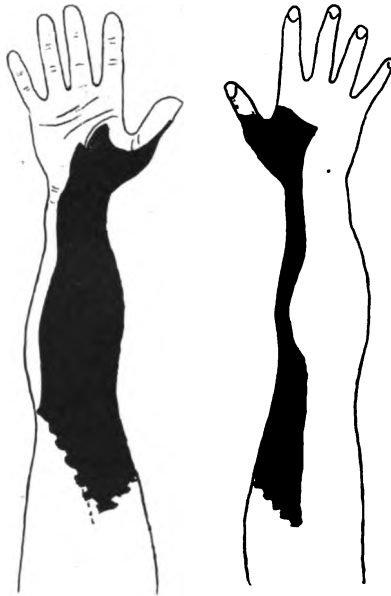


Fig. 19.—Residual sensibility to prick pain of the musculocutaneous nerve.

had distinctly increased. If Figure 3, *b*, page 136, represents the sensory chart of this case after operation, a considerable area sensitive to pain within the area of anesthesia still requires explanation.

To have an increase of analgesia following resection and suture is not sufficient to prove the above contention. The whole area of anatomic sensory supply of a nerve must again become analgesic. That this did not occur even in Head and Sherren's cases is shown by Case 28, page 322. In a combined ulnar and median lesion the ends of the median nerve were found separated, but the ends of the ulnar

13. Head and Sherren: *Brain* 28:108, 1905.

were connected by a thin strand of tissues. Following resection and suture, a considerable increase in the area insensitive to prick resulted. This increase occurred in the distribution of the ulnar nerve and may represent very well the change to a complete from an incomplete lesion. But even here the analgesia did not coincide with the anesthesia. The radial portion of the palm, the thenar eminence and the thumb remained sensitive to pain. This area, therefore, was the area of overlap of some other nerve to the median. Return of sensation in this area may be due to overlap, and never should have been considered in estimating the regeneration of this nerve.

My cases definitely show that, following resection and suture when sensibility to pain is present in an area of overlap, although some change in the outline of this area occurs, in general the area remains the same. At times the borders show some increase in analgesia; much more frequently they show a shrinkage of the analgesia. Slight changes in the borders of an area of analgesia cannot be used in arriving at a hard and fast conclusion. Frequently these borders change in an astonishing manner for pain produced by higher degrees of pressure by a sharp point not sufficient to produce pressure pain.

The laws governing dual innervation have not been clearly ascertained. What effect, if any, the handling of nerves or freshening of their ends may have on inhibition is unknown. In my opinion, another fact in support of the statement that return of sensation in an area of possible overlap cannot be ascribed to the regeneration of a nerve is that this area is not generally changed by resection and suture of a severed nerve.

The conditions necessary to study profitably the effect of resection and suture of nerves on return of sensibility to pain are: First, that the nerve ends be separated, and, second, that the examination subsequent to the operation be made within the period of time ascribed to the return of protopathic sense.

Some difficulty is encountered in meeting the second condition inasmuch as frequently the wide separation of the ends of the nerves makes it necessary to place the extremity in a position which will permit approximation, and to fix it in such a position by means of a case. This often prevents an examination before six weeks have elapsed. None of the cases here reported were examined later than fifty days after operation, one in less than fifteen days. Although some objection may be made to the cases examined over forty-five days after operation on the grounds of beginning return of protopathic sense due to regeneration, the similarity of the areas unaffected by operation in cases examined under forty-five days and those between

forty-five and fifty, coupled with the facts that the ends of the nerves were separated in all of these cases, makes it reasonable to admit them into the group.

To describe again the areas sensitive to pin prick in the lesions examined, or to attempt by description to show the sensory changes following operation, is needless. They are clearly indicated by

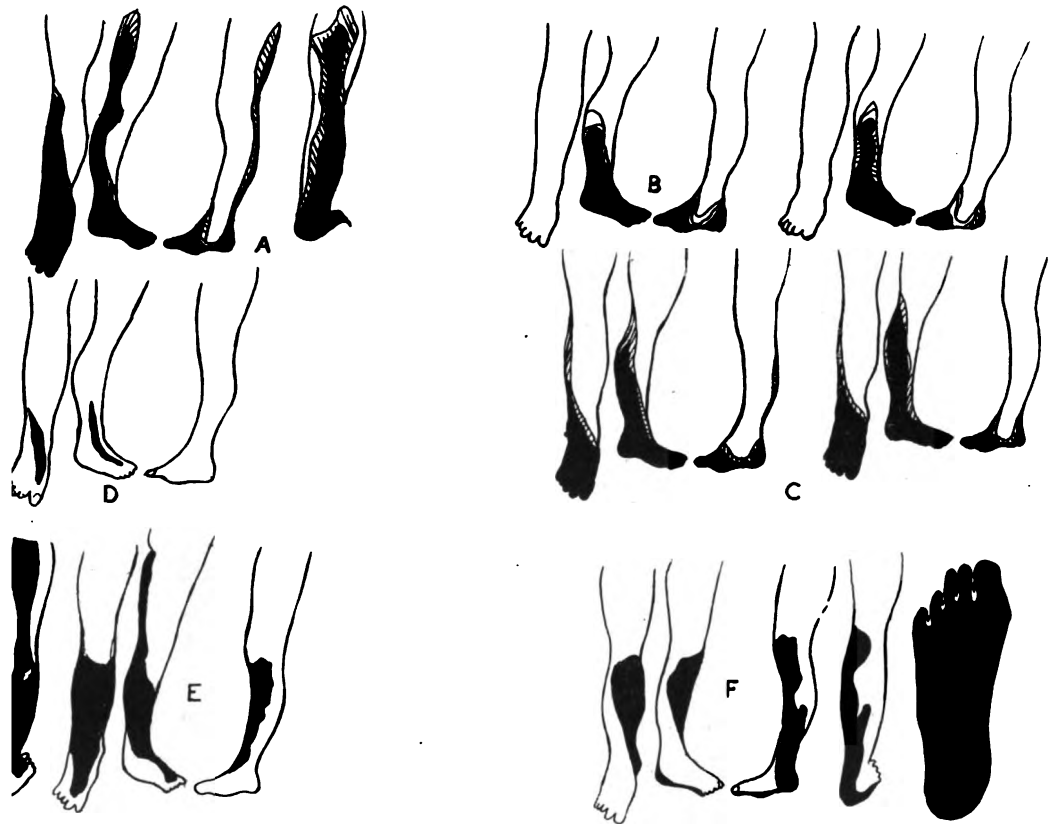


Fig. 20.—Sensory changes of combined lesions of internal saphenous and internal popliteal nerves, *f*; small sciatic, external popliteal and internal saphenous and sciatic nerve lesions from which the residual sensibility of the external and internal popliteal nerves was obtained.

Figures 13 and 14. It is sufficient to state that the following nerves were studied: ulnar, examined forty-two days after operation, Figure 13 *a*; median, eight days after operation, Figure 13 *f*; ulnar and median, forty-five, thirty-six, forty, forty-six, forty-eight and fourteen days after operation (Fig. 13 *b, c, d, e, g, h*); external popliteal, forty-eight, thirty-six, twenty and twenty-six days after operation, Figure 14 *a, b, c, d*; sciatic, fifty and thirty-six days after operation, Figure 14 *f, h*.

3. RESIDUAL SENSIBILITY

If we assume the relatively early return of sensibility to pin prick to be due to overlap it becomes possible by the method of residual sensibility to outline the borders of overlap of the various peripheral nerves.

The method of residual sensibility is based on the assumption that following section of a given nerve, the area of skin, in its anatomic distribution in which sensation remains, is subserved by the intact adjoining nerves distributed to that area. For example, four nerves supply the palmar surface of the hand: the ulnar, median, musculocutaneous and radial. If two—the ulnar and median—are severed, what sensibility remains is subserved by the musculocutaneous and radial. If then the borders of the musculocutaneous be determined, that which remains is radial.

In employing this method certain precautions must be observed. For example, we cannot take the outer border of the analgesia on the dorsal surface of the hand in an ulnar section to be any part of the border of the overlap of the median unless we may observe the effect of a combined ulnar and musculospiral so that the overlap of the latter nerve be not included. Similarly, we cannot outline the border of the overlap of the median on to the radial unless we have had a combined lesion of the ulnar and radial to indicate the distribution of the ulnar; or the overlap of the radial to the median on the palm unless we have had a combined median and musculocutaneous lesion, or the overlap of the internal popliteal to the external popliteal unless we have had a combined lesion of the external popliteal with the internal saphenous. The necessity for these combinations reduces the number of cases available for conclusions to a very few. As a result the areas of overlap as outlined probably were smaller than the real overlap. However, the extent of overlap was sufficiently large to prove my contention that it is within such an area that return of sensibility to pin prick occurs soon after injury of peripheral nerves.

Owing to this clinical lack of certain combination injuries, I have been unable to determine all the overlap areas of some of the nerves investigated.

It is hardly necessary to state that the cases studied must have nerves recently resected or be examined prior to an operation which reveals the ends of the nerves separated.

In illustrating the areas of overlap, the space between the borders of the overlapping to adjacent nerves has been blocked out with black. The black area, therefore, represents the total supply to pain of the various nerves studied. The area of actual overlap would be that part of the total sensory supply to pain which extends beyond the

accepted sensory limit of the adjacent nerves. The restrictions of the methods necessary to obtain these areas are responsible for an indicated area of total supply, which in some instances is smaller than is actually present, as may be seen in the case of the outer border of the ulnar on the dorsal surface of the hand and the inner border of the external popliteal on the back of the leg (Figs. 17 and 21).

The area of total supply to pain of the ulnar nerve was obtained by the method of residual sensibility from a median lesion, an internal cutaneous lesion and a combined median and radial (Fig. 15, *b, g, m*). It occupies the ulnar portion of the palm to a line which is a continuation of the ulnar border of the abducted index finger, the palmar surface of the fingers except the terminal phalanx and one-third of the ulnar part of the second phalanx of the middle finger. On the

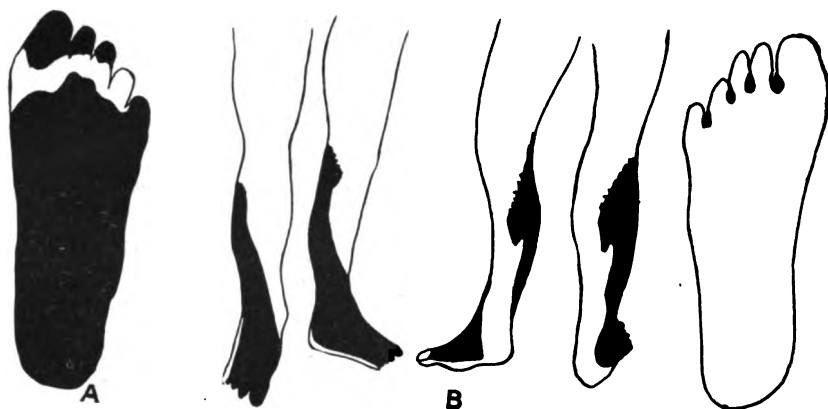


Fig. 21.—Residual sensibility to prick pain of external popliteal nerve, *b*; sensory changes in an uncertified case of complete interruption of the internal popliteal, *a*.

dorsal surface it occupies the ring, little and more than the ulnar half of the proximal one-and-a-half phalanges of the middle finger, the dorsum of the hand to the radial border of the fourth metacarpal bone, ending proximally one inch above the wrist (Fig. 16).

The area of the median nerve was obtained from an ulnar and internal cutaneous lesion, a radial, a combined radial and median lesion and cases of combined radial and ulnar lesions (Fig. 15, *h, j, m*). The inner border on the palmar surface was obtained by the method of residual sensibility from an ulnar and internal cutaneous lesion. On the dorsal surface I was compelled to employ another method, as the cases of combined ulnar and radial lesions were too recent to have had return of prick pain due to overlap. The border of overlap of the musculocutaneous to the radial was obtained by means of residual

sensibility in a case of combined radial and median. Inasmuch as the radial has no isolated supply to prick pain, this border separates the musculocutaneous from the median overlap. I, therefore, used this border as the proximal border of the median overlap to the radial nerve. We were supported in doing this in such cases as showed an area of analgesia between the areas of overlap of the median and musculocutaneous nerves (Fig. 15). Part of the inner border of the overlap on the dorsum of the hand is hypothetical and shown as a rough border (Fig. 17).

The total supply of the musculospiral nerve was obtained from cases of combined ulnar and median lesions, a case of combined ulnar, median and musculocutaneous lesions and a case of combined ulnar and internal cutaneous lesions (Fig. 15, *a, d, e, f, h, i, k, l, m, n*). I found an overlap on to the palm to an extent heretofore undescribed. In median nerve lesions the sensibility to pain in the palm has frequently been ascribed to ulnar overlap. But Athanassio Benisty⁹ recognized the importance of the musculospiral and the musculocutaneous nerves in this condition.

The area of overlap on the palm of the musculospiral nerve extends over the radial part of one-and-a-half phalanges of the index finger, the radial part of the proximal phalanx of the middle finger, and the web between the middle and ring fingers, all of that part of the hand external to a line continuous with the radial border of the middle finger. Internally, it extends from the middle of this line to the middle of the base of the first phalanx of the ring finger and proximally to the middle of the outer surface of the wrist, from which point the border extends in a line to a point one inch proximal to the base of the metacarpal bone of the thumb on the radial border of the wrist. This area occupies the entire dorsal surface of the hand, with the exception of a strip one half the width of the little finger on the ulnar border, the little finger, the distal two phalanges of the ring finger, most of the distal two phalanges of the middle finger and a little more than the distal phalanx of the index finger. The area on the forearm need not be described (Fig. 18).

The inner border of the pain area of the musculocutaneous nerve on the anterior surface of the forearm was obtained from the residual sensibility following section of the internal cutaneous nerve; the inner border on the dorsal surface of the forearm, from a musculospiral division. The distal border on the palm was obtained from radial lesions, a combined radial and median lesion, and from lines obtained in combined ulnar and median lesions where an area of analgesia existed between the areas of overlap of the musculospiral and the musculocutaneous (Fig. 15, *e, f, k*). On the dorsal surface of the

hand combined sections of ulnar, radial and median and a case of combined radial and median were employed (Fig. 11, *a, b, c, f*; Fig. 15, *m, h, j*). The area of total sensory supply to pain of this nerve can be better appreciated by viewing the illustration than by description (Fig. 19). The proximal limits of both the musculocutaneous and musculospiral nerves are hypothetical.

I was fortunate in obtaining two cases from which the overlap of the internal and external popliteal nerves could be obtained according to the method of residual sensation. One was the case in which the internal saphenous and internal popliteal nerves were injected with alcohol for causalgia, producing anesthesia, the residual sensibility about which permitted the outlining of the total supply for pain of the



Fig. 22.—Residual sensibility to prick pain of the internal popliteal nerve.

external popliteal nerve (Fig. 20, *f*). The upper border of this area is hypothetical and merges on the outer surface with the external cutaneous, on the posterior surface with the small sciatic and on the inner side with the obturator nerve.

The overlap on the sole seen in Figure 21 *b* is probably smaller than that which actually exists, as may be seen from the presence of sensibility to pain in the blank area of the sole in a case of complete interruption of the internal popliteal nerve, a case which, because it was not certified by operation, is not included in my series (Fig. 21, *a*).

The area of total supply for pain of the internal popliteal nerve was obtained from the residual sensibility in a case of a combined lesion of the small sciatic, the internal saphenous and the external

popliteal nerves, and a case of external popliteal section (Fig. 20, *d, e*). The upper border of this area is hypothetical and merges with the borders of the small sciatic and obturator nerves (Fig. 22).

The area of total pain supply of the internal saphenous nerve was obtained from the residual sensibility of a combined lesion of the small and great sciatic and cases of sciatic section (Fig. 20, *a, b, c*). The upper border here is likewise hypothetical, merging with the borders of the anterior crural, the external cutaneous and the obturator nerves (Fig. 23).

THEORETICAL CONSIDERATIONS

The evidence adduced above to show that the early return to sensibility to pain cannot be interpreted as a sign of nerve regeneration, but probably occurs as the result of nerve overlap, does not invalidate that part of the theory of Head and his co-workers which relates to the existence of a distinct system of fibers for epicritic and for protopathic sensibility. The question of the existence of these two systems can probably be settled only by psycho-physical research.

Boring⁷ defines in part the sensory changes following section of a sensory nerve as follows: "We have seen that in the outer zone of the affected region there is at any sensory spot (the region stimulated by a punctiform stimulus) a partial loss of sensitivity. Since the partial loss comes as the result of the complete severance of certain fibers, we may presume that the partial function remaining occurs by way of other fibers which have not been cut (principle of multiple innervation of a sensory spot). These must come from an adjacent nerve."

I do not believe that any hypothesis of dual innervation is immutable. The probability that the return of so-called protopathic sense before the return of epicritic sense is due to nerve overlap and not to a temporal dissociation of protopathic and epicritic sensibilities attacks one part of the hypothesis of Head and his co-workers. This emphasizes the necessity for review of the theories of dual innervation and for that reason I shall quote freely from Boring (p. 89):

Suppose, now, that we have a spot which is innervated primarily from the nerve *P*, but which is also innervated secondarily by fibers from the nerve *S*. Let such a spot be *X*, Figure 97, *a*. Since *P* and *S* are from adjacent nerves, they will be projected upon adjacent regions at the center; and, since *P* is primary and *S* secondary, the corresponding excitations, *p* and *s*, will be unequal — that is to say, *p* will be greater than *s* (Fig. 97, *a*). Thus *s* may be subliminal and *p* supraliminal, so that *p* alone is adequate to sensation. We may assume this to be the normal state of affairs; namely, that the primary fibers are alone effective, and that their effect is always partially decreased by the secondary effect (for the secondary fibers, projected upon a different region, would tend to inhibit the primary: . . .).

Now let the nerve P be cut (Fig. 97, b). At once the inhibiting effect of p upon s is removed and s is increased. It may, however, still remain subliminal. Thus it appears that, even in an area of complete sensory loss, there may be fibers from adjacent nerves which tend to function, but which are not adequate to a supraliminal excitation.

As time goes on, the secondary fibers would tend to take on the function of the primary fibers (principle of vicarious function). The many cases of gradual recovery from permanent lesion have shown that, whenever possible, an interrupted function is taken over by other elements which are spatially or functionally connected, and that the vicarious assumption is not immediate but gradual—by practice. Thus, as the S —fibers became more “practiced,” the s —excitation would become first supraliminal (Fig. 97, c), and then, since there is no p —excitation to inhibit it, it might become more intense than the p —excitation was originally (Fig. 97, d).



Fig. 23.—Residual sensibility to prick pain of internal saphenous nerve.

The next stage would occur when the P —fibers began again to make functional connection (Fig. 97 e). The s —excitation would be diminished by the small, subliminal p —excitation. The return of functional adequacy to P would doubtless be gradual, but eventually p would become effective for sensation, and two sensations (Fig. 97, f) would be felt. If now P is so situated as to form better or more connections with the sensory spot, x , than is S , then it will come to dominate S in its demand upon central energy; and S , now less effective, will undergo the functional decay, which is the converse of “practice” in vicarious functioning. Thus, from the conditions of Figure 97, f , the original normal conditions of Figure 97, a , would finally be recovered.

The significance of the figures on the introspective side requires only a word. At first, stimulation of x gives rise to a single sensation, normally localized (Fig. 97, a). Then the nerve is cut and there is no sensation (Fig. 97, b). Then stimulation of x produces a weak sensation, remotely referred with regard to the localization of the original sensation (Fig. 97, c). The sensation becomes abnormally intense (Fig. 97, d); then decreases (Fig. 97, e); and then

is supplemented by another sensation referred to the original point (Fig. 97, *f*). Finally, the normal condition recurs (Fig. 97, *a*). Such would be a course of returning sensibility, although it is not necessarily the typical course.

On our hypothesis it is not possible that all the fibers of any one nerve are projected upon the same central area. In dividing a nerve, we must divide both primary and secondary fibers for many sensory spots. Such a condition is diagrammed in Figure 98. The normal excitation (Fig. 98, *a*; compare with Fig. 97, *a*) is entirely abolished by the section of both the fibers *P* and *S* (Fig. 98, *b*). Since the primary fibers are probably in a better position to make functional connection than are the secondary, they would be the first to become effectively connected; and we should have the subsequent stages of Figures 98, *c* and 98, *d*. If the secondary fibers did not return rapidly to function, the *p*-excitation, relieved of their inhibiting effect, might rise above normal (Fig.

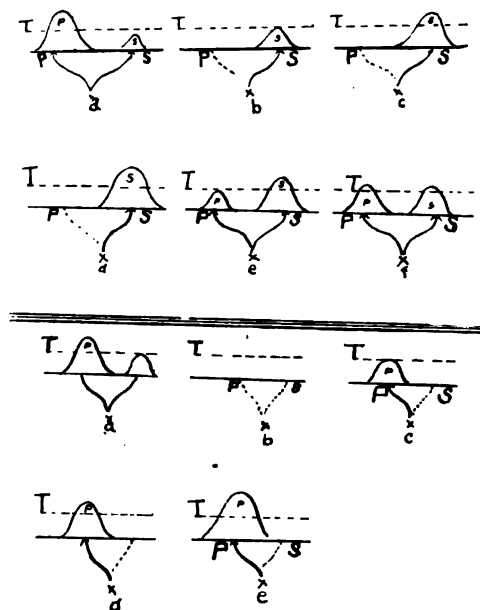


Fig. 24 (Figs. 97 and 98, Boring).—Successive changes with respect to intensity and reference of sensation, dependent on the stimulation of a single point, dually innervated after the division of one (Fig. 97) or both (Fig. 98) of the innervating fibers (see text). Compare Figures 94-96. Dually innervated point on the skin, *x*; primary innervating fiber, *P*; secondary innervating fiber, *S*; corresponding central excitations, *p*, *s*; central threshold, *T*.

98, *e*). The subsequent return of the *S*—fibers would, however, eventually bring about the normal state again (Fig. 98, *a*). Here, then, we have a case in which recovering sensibility passes through a stage of abnormal intensity, without, however, being abnormally localized.

Results of my investigation permit criticism of only that part of the theory of Head and his co-workers dealing with the temporal dissociation of epicritic and protopathic sensibility.

CONCLUSIONS

1. The area of prick pain supplied exclusively by an individual nerve is far less than the accepted sensory distribution of that nerve.
2. The area between the border of exclusive supply of prick pain of an individual nerve and the border of its accepted sensory supply constitutes the area of algesic nerve overlap.
3. When nerves serving adjacent areas are severed, sensibility to prick pain between these areas is not present after injury, nor does it return before the sense of touch.
4. When a region in the area of sensory distribution of a severed peripheral nerve is sensitive to prick pain, and this region is adjacent to another nerve area, if this second nerve be severed, complete analgesia results in the previous sensitive region.
5. When sensibility to prick pain is present or returns in the area of possible overlap on to the sensory distribution of a severed nerve, subsequent resection and suture of this nerve does not change the general extent of this sensitive area, although the borders may at times be slightly enlarged or diminished. That is, the pain sense returned or present before the operation was not due to partial regeneration.
6. The laws governing the assumption of function by nerves adjacent to a severed nerve are unknown.
7. Handling and resection and suture of previously divided nerves changes the condition governing the function of overlapping nerves, often initiating greater function.
8. Evidence of the assumption of function by nerves adjacent to a severed nerve is not present immediately following the nerve injury, but gradually shows itself at a later date.
9. The early return of sense of prick pain before the return of sense of touch is not due to temporal dissociation of epicritic and protopathic sensibilities, but is due to the assumption of function by adjacent overlapping nerves.
10. The areas of overlap may be determined with fair accuracy and the early return of sense of prick pain in those areas cannot be interpreted as a sign of regeneration of the divided nerve.
11. The changes in prick pain following division of a single nerve are not a safe basis for conclusions regarding regeneration of that nerve.
12. Only when a group of nerves is divided at the same time can the studies of sensation be used in the interpretation of regeneration of these nerves. Under these conditions only that part of the analgesic

area may profitably be studied which is removed from the effect of overlap from adjacent nerves. On the other hand, if return to sensibility to prick pain occurs on the border of an uninjured adjacent nerve, this return to sensibility does not indicate regeneration of a nerve.

13. Return of sensibility to prick pain can be used clinically for the determination of nerve regeneration only when it is accompanied by return of tactile sense or when it occurs outside the area of possible overlap of adjacent nerves.

In conclusion, I wish to express my thanks to Lieut.-Col. Dean D. Lewis, whose encouragement and interest made this work possible, and my gratitude to Capt. Wesley Gatewood, who, by his industry and enthusiasm, contributed no small part of the matter contained in this communication.

Abstracts from Current Literature

ASCESSI E TUMORI DELL'ENCEFALO. Osservazioni Cliniche Ed, Anatomopatologiche del dott. G. MINGAZZINI, Ordinario di Neuropatologia nell Regia Università di Roma, Riv. di patol nerv. **24**: Parts 3-4 and 5-8, 1919.

The author has written an extensive article, which appears in two numbers, and merits this rather long review. He mentions the numerous diagnostic difficulties, particularly as to localization of brain abscesses and tumors, and ascribes the poor surgical results which surgeons obtain in these cases to the facts that first, the neurologist is unable to give always accurate localization of the process and second, the diagnosis is not made early enough. In brain abscesses, even when evacuation is possible, death will occur because of the edema which involves vital areas or because of the "marasmus" which has begun. We do not possess unfailing guides to differentiate cortical from sub-cortical tumors. Unfortunately, exactly this differential point is necessary before the operator can use surgery with success. It is not enough to tell a surgeon that the tumor is frontal or temporal, for these areas are too extensive to allow complete exposure at operation. These cases must be minutely studied and more accurate localization arrived at. In tumors and abscesses of the cerebellum, however, this is as yet impossible and very wide exposures are advised by Duret and Murri. By this method one is able to explore for tumors with the finger, but it is only by means of exploratory puncture in various directions that one can locate the site of abscesses in the cerebellum. Murri states that the number, progression and character of the symptoms produced by tumors of the frontal and temporal lobes are so varied that no one can lay down fixed rules. Nevertheless, the author believes that constant attempts should be made to study the symptoms, and thus definitely localize the process.

The author's material consisted of four abscesses of the cerebellum and eight brain tumors, three of which were in the posterior fossa, three in the middle and two in the frontocentral fossa. In localizing a cerebellar abscess, the difficulties are increased because of the almost invariable presence of mental confusion which interferes with the delicate tests necessitating good cooperation or because, as often happens, an abscess may occur in a patient who has double otitis media. With these preliminary remarks, the author describes in detail his four cases of cerebellar abscess. In order to know exactly how he observes his material, the first case is reported in some detail.

The patient had an abscess in the right cerebellar hemisphere, was operated on and died. Syphilis was denied. There was a history of acute otitis media, with a cessation of discharge from the right ear; a few days later he had headache (occipital), more severe on the right side and when standing. The patient became rapidly semistuporous, but on being transferred from the ear department to the author's service, he brightened up enough to give his history. The temperature had always been subnormal. On Feb. 28, 1918, four weeks after the onset with headache, there was rigidity of the neck, especially on movement to the right; pressure on both jugulars did not cause pain; there was limitation of mobility of the right eye to the right; there was no interference with other movements. There was a slow nystagmus, especially on

looking to the left, a mild right facial weakness, but no limitation of tongue movements or deviations. The patient was not able to hold his hands upright in the attitude of a person who is being sworn, both falling rapidly; the right extremity oscillated considerably as it fell. This attitude is one which the author mentions as a test for paresis in the upper extremities, and he apparently thinks it is an important test. There was diminished resistance to passive movements on the right side, and both lower limbs could not be held raised in the air. There was decided weakness, therefore, in both the upper and lower right extremities, but active movements were well made. Both pupils were widely dilated, and light reaction was absent. The tendon reflexes on the right side were much diminished, but were quite active on the left. There was plantar flexion in both big toes; abdominals were active, and the right corneal was diminished. Percussion of the skull was painful in the right occipital region; pain, temperature and tactile sensibility were unchanged; the finger to nose test on the right side was badly done, but *asynergia* could not be tested for, on account of the patient's mental condition. The patient could stand alone only on a wide base with his feet widely apart. He could walk if supported, swaying to the right or left; if not held, he fell, usually toward the left. He lay on the right side, in bed, with head bent backward; when he arose the headache was worse; he past pointed spontaneously to the right with the right finger; sense of taste and smell were not tested. There was a bitemporal pallor of both disks; pulse, 60; urine, negative. The patient cried, shrieked and was semi-stupid. He was oriented for time, place and person. Attention was poor and perception not good. Lumbar puncture gave a few drops of turbid, bloody fluid under slight pressure. No fluid could be aspirated with a syringe.

The patient died Feb. 28, 1918, and a necropsy was immediately done. The right cerebellar hemisphere was found much swollen, the lobus quadratus soft in consistency and only when frontal sections were made, did a fetid pus escape from the ventral surface of the hemisphere, leaving a cavity which took in the subcortical area corresponding to the lobus quadratus and lobus semilunar posterior. There were two other necropsies, making a total of three of four cases of cerebellar abscesses and in the fourth, the diagnosis was verified at operation.

DISCUSSION

Oppenheim and Cassirer are referred to by the author as authorities for the statement that abscesses in the posterior fossa cause the most intense occipital and nuchal pain, but occasionally frontal and rarely parietal and temporal headaches occur. In the author's Case 4 (abscess of the anterolateral left hemisphere of the cerebellum) headache was in the left frontal and temporo-parietal region. In his Cases 2 and 3 headache was not localizable but constant (abscesses of antero-medial and antero-inferior portions of cerebellar hemisphere). In Case 1, headache was worse on the right side and in erect posture (abscess of dorsolateral portion of the right cerebellar hemisphere). From the author's cases, it would appear that headache is diffuse if the abscess is central and inferior, but is worse in occipital region on the same side as location of abscess. Mingazzini warns against using pain as a localizing sign without careful consideration of other symptoms. Oppenheim and others assert that tenderness to percussion and pressure is worse when applied to the same side as the lesion and usually corresponds to the site of maximum headache. This was true in the author's cases. Mingazzini also confirms Oppenheim's finding of dizziness and vomiting as early symptoms, these occur-

ring in all four cases, most marked, however, in Cases 1 and 2. Oppenheim finds that optic neuritis (blurring hyperemia, loss of outlines) occurs in cerebellar abscess frequently; not infrequently a papilledema may occur, always worse on the side of the abscess. The author says, however, that a number of cases occur which never show fundus changes at any time in their course (his Cases 2 and 4 are cited). In the other two cases there was bitemporal pallor in one, optic neuritis in the left eye and papilledema in the right eye (same side as abscess) in the other case. Acuity of vision was retained in all four cases, but blindness may occur. All his cases showed the following mental signs: clouding of the sensorium, poor perception and attention and in one case disorientation for time. In Case 1, there was early stupor. Only in episodes does one see restlessness and excitement. The pulse is usually slow.

None of his cases had cerebellar seizures (attacks of vertigo, nystagmus, very severe headache and paresis). The author's cases confirm the statement that in cerebellar abscess, nystagmus is usually toward the side of the abscess. In Case 4, the movement was slow, rotary and appeared only when the patient looked toward the affected side. In Case 1, however, this appeared only when the patient looked away from the side with the abscess. There was paralysis of gaze or of one sixth nerve alone, usually homolateral. In Case 2, the sixth pair was affected, probably because in this case the pus tended to collect in the base in the median line. In Cases 3 and 4, there was a paralysis of the twelfth cranial nerve, the tongue deviated toward the side of the abscess in one and away from it in the other. The author states that it is difficult to say whether this paralysis is due to compression of the nucleus of the twelfth nerve in the medulla or of the supranuclear pathways, but thinks it is probably due to both; that is why the tongue deviates sometimes toward and sometimes away from the lesion. The seventh pair of nerves in the author's cases were paralyzed on the same side as the abscess, the explanation being that pressure is exerted by the abscess at the base as the nerve courses there. The author does not believe that suppurative processes in the internal ear cause the seventh nerve palsies. Ataxia and adiadokocinesis was present on the same side as the lesion in two of his cases; but in Case 1, when the patient walked, there was a tendency to fall to the side opposite to the lesion. Many authors state that this symptom is always toward the side of the lesion, and this is usually true; but in this case the vermis was undoubtedly involved and therefore the cerebellospinal pathways were interfered with, causing the swaying toward the opposite side, but in Case 2, the underlying pyramidal tract was injured causing the tendency to fall toward the side of the lesion. In only two cases was ataxia present, but in explaining this, the author points out that weakness often interferes with tests for coordination; also that the mental condition interferes with the sensory examination. Nevertheless, he was able to find hypesthesia in the distribution of the right fifth nerve in Case 2, and in Case 4, hypesthesia including pressure and vibratory sensibility and some degree of astereognosis on the same side as the abscess. He never found hyperesthesia. In Case 4, the abscess undoubtedly pressed on the tegmentum pontis because of its situation in the left anterolateral hemisphere of the cerebellum. Only in one case (2) was there any Babinski phenomena (fanning). In Case 3 (abscess on antero-inferior surface), the patellar and tendo-achillis reflexes were entirely abolished on both sides and in Case 2, much diminished on the side of the abscess. Whether this rule of diminished reflexes holds good, depends entirely on the degree of involvement of the pyramidal tracts. The corneal reflex was diminished in only two cases, in

both occurring on the same side as the abscess. The size of the pupils follows no definite rule, and is therefore not helpful in localizing. The mydriasis or myosis depends on the degree of pressure endured by the third cranial pair of nerves; double ptosis was present in one case. Exophthalmus, depending on hydrocephalus, occurred on the same side as the abscess in one case. Dysarthria from pressure on the pons and medulla is rarely seen, but was present in Case 4. Attempts to aid in localization by attention to errors of pointing and past pointing should be discounted by the difficulties of cooperation because of the patients' mental condition, weakness and possible loss of position sense. In two of the author's cases, deviations in past pointing were never constant. In Case 1, the patient past pointed to the right; the lesion was in the right dorsolateral hemisphere. This does not seem to bear out Bárány's claim, that past pointing to the right in the right upper extremity is localized in the inferior and superior semilunar lobes of the cerebellum. They were intact in this case.

All four patients died. Three were operated on, the pus was evacuated in two, and not found at operation in one. The delay was fatal in all cases. In Case 4, the abscess drained well and improvement occurred, but death followed due to the formation of another abscess. According to Oppenheim and Cassirer, this often happens—the abscess is evacuated but later another develops. The author advises puncturing the cerebellum in many directions until pus is found. In one of his cases, at first operation punctures failed to find pus; a few days later, the needle being introduced in other directions, much pus was found.

The most constant signs of cerebellar abscesses are: mental clouding, headache, rigidity of the neck, vertigo, vomiting and changes in the fundi. Less frequently there is cerebellar ataxia, fever and marasmus. The localizing signs are usually found on the same side as the abscess: pain on percussion, hemi-ataxia which is static or dynamic, adiadokocinesis, hemihypesthesia, areflexia of cornea; less frequently, myosis, sluggish reaction of pupils to light, optic neuritis, error of past pointing and change in the deep tendon reflexes. The latter, however, together with deviations of the tongue and the position in which the patient lies, when in bed, cannot be used as aids to localization.

The author then presents his cases of tumor of the brain. The first one—Case 5—is a case of tumor of the right cerebellar hemisphere, tegmentum pontis, et bulbi. This case was particularly difficult because for a long time there was a justified suspicion that it might be a case of cerebellar abscess. The boy, 11 years of age, had had chronic otitis media for several years until May, 1917, when the discharge from the right ear suddenly stopped; then began headache, vomiting, vertigo (objects whirled from left to right). The headache was most severe at the vertex and left parietal region. There were slight elevations of temperature (37.2) daily, with chills. In June the patient had diplopia, gradual loss of vision and loss of hearing in the right ear, together with disturbances of gait. The pulse was 66, lumbar puncture showed a fluid under great pressure, negative "sediment and Nonne, 1 per cent. albumin and no coagulation." The findings were: headache, vomiting, dizziness, fever, mental dulling, papilledema, exophthalmos in the right eye, paralysis of all the cranial nerves, including the fifth to the twelfth, hypesthesia and hypothermalgesia in the faciobrachial distribution with loss of vibration sense in four toes of the right foot, hypotonia and static ataxia of all the extremities, restriction of the right visual field, diminution of the

sense of smell, diminished reaction of both pupils to light, absent patellars and tendo-achillis reflexes diminished, areflexia of the cornea and spontaneous past pointing to the right. The patient had a tendency to lie on his left side. Cranial percussion was more painful in the left parietal occipital region; there was bilateral nystagmus and cerebellar gait with a tendency to fall toward the left.

At necropsy a white swelling was found in the middle of the gray matter of the right cerebellar hemisphere which was continued downward into the right half of the pons where it was almost impossible to make out the formation of the tegmentum, particularly of the lemniscus and the formatio reticularis. In discussing this case, the author mentions the fact that the location of the process in the cerebellum, pons, and medulla of the right side was suspected from the beginning, despite the fact that there was no paralysis of gaze. But he agrees with Murri that this symptom is not constant in tumors of the pons because alternating palsies of the eye muscles occur which are sufficient in themselves for intrapontile localization. Cerebellar abscess was suspected chiefly on account of the sudden cessation of the discharge from the right ear and the slight elevation of temperature, with great loss in weight. The author points out that while hypotonia existed in the right extremity, the patient in walking fell toward the left; this he explains by the displacement of the vermis obliquely toward the right, injuring in this way the pathways proceeding from the right cerebellar hemisphere and thus interfering with the tone and taxis of the right side. In order to explain the right faciobrachial sensory changes, an invasion of the left side of the tegmentum pontis must be supposed, involving a great part of the trigeminus and the lemniscus on that side. Particular mention is made of the fact that the patient preferred to lie on the left side, and suffered most pain in the left parieto-occipital region on the side opposite the tumor. While this is not usual, Bruns explains it by pointing out that cranial percussion is most painful in tumors of the cerebellum associated with marked hydrocephalus, while diffuse pain is most common with tumors in the cerebellar hemispheres near or on the cortex. The author explains the presence of a pain on the side opposite the lesion by ascribing it to the compression exerted on the healthy hemisphere and its dura by the diseased side.

Concerning his sixth case, which was a tumor of the tegmentum pontis on the left side, the author says that the progression of the symptoms suggested at first a neoplasm of the cerebrum even though there were no disturbances of the fundi or of vision. The dominant symptoms at first were: paralysis of gaze toward the left, weakness of the left internal rectus and of the seventh nerve, and on the right, an alternating paralysis of the extremities with hypesthesia of both superficial and deep sensations. The latter symptoms made it probable that the tumor was situated in the anterior portion of the left tegmentum pontis, and that it extended forward toward the pyramid. The absence of the left corneal reflex and the spontaneous error of past pointing of the right hand to the right made one suspect that the tumor had begun in the left posterior fossa and then had compressed the right cerebellum. Later, however, when adiadokocinesis, ataxia, cerebellar gait, etc., had made their appearance, it was suspected that not only the tegmentum, but also the brachium pontis of the left side had been invaded. The author believes that the various forms of sensibility run through the tegmentum pontis in separate groups—the stereognostic sense fibers through the middle of the lemniscus, tactile sensibility through the posterior zone of the lemniscus close to the

formatio reticularis, while the pain fibers and those of heat and cold seem to run through the formatio.

In his seventh case, which was a tumor of the left cerebellopontile angle, the author discusses the characteristic triad of this syndrome—disturbance of function of the fifth and eighth nerves with nystagmus. In the author's case there was also involvement of the third and fourth nerves. In tumors of the pons itself, the eighth nerve is less often involved and usually much later than in tumors at the angle. Also, papilledema is less frequent and the course of the disease more rapid. Some difficulties in the diagnosis of the author's case were found on account of the presence of weakness and atonia on the right side opposite to the pareses of the cranial nerves, such as often happens in pontile tumors, but the unilateral character and the early appearance of cochlear and vestibular symptoms, together with the more marked papilledema on the left side, localized the tumor. The exophthalmos was also more prominent on this side. Concerning the cochlear and vestibular functions, Mingazzini refers to many authorities who agree with him that while the cochlear functions are never destroyed so that complete loss of hearing occurs, yet vestibular excitability of the affected side may be completely lost and diminished on the sound side. Three cases are referred to in which the vestibular functions were excitable also on the side of the tumor. The author believes that a diagnosis of cerebellopontile angle tumor can be made whenever there are disturbances of function of the fifth and eighth nerves, paralysis of the sixth and seventh, nystagmus, paralysis of lateral gaze, exophthalmos, hypogeusia and the tendency of the body to fall toward the suspected side, with increase of cranial percussion pain on the affected side. Location of weakness, atonia and disturbance of reflexes are considered of little importance because in some cases the dentate nucleus is irritated, while in others it is destroyed, which accounts for the difference in symptoms. In tumors of the cerebellum, however, the side on which disturbances of the cranial nerves are found is not necessarily the side of the lesion because the tumor may be exerting its pressure in an oblique contralateral manner, compressing the other side. Particular attention must be paid to the progression and history of the onset of the various symptoms. The author agrees with Cushing in laying great stress on paralysis of the peripheral type of the seventh nerve as an important localizing symptom of tumors of the angle. This may exist alone for a long time before other symptoms occur. It is possible that only one branch of the seventh nerve may be involved. The author lays great stress on this point.

In these three cases (5 to 7) the author points out the paucity of mental disturbance in tumors of the posterior fossa as contrasted with the more marked mental disturbance in tumors of other parts of the brain. In his case of angle tumor the author also directs attention to the fact that there was great variation in the spinal fluid findings, not only as to pressure, but some cases have also been described in which the Wassermann and globulin reaction was positive. He says that a positive Wassermann is frequently found in nonsyphilitic tumors of this region.

Case 8 was a tumor of the median portion of the fovea cerebrealis media dextra. The patient began with a progressively increasing headache, dimness of vision, vomiting and hiccup. The roentgen ray disclosed disintegration of the sella. On the right side there was almost complete ophthalmoplegia, hypesthesia of the face, mydriasis, painful percussion and blindness. On the left side, there was weakness of the extremities, increase of the deep

tendon reflexes of the lower extremity and a double atrophy, more marked on the right side. The only focal sign of brain tumor was an incomplete superior alternating type of paralysis. This was the only sign which at first led to the belief that the tumor was in the base of the pes pedunculi on the right side or in the middle fossa. Against the first supposition was the poorly marked paralysis of the extremities and the involvement of the fifth nerve on the right side. It seemed more probable that the tumor had begun in the medial portion of the middle fossa on the right side near the sella with a tendency to penetrate the sphenosphenoidal fissure. This would explain the unilateral syndrome which was present. The roentgen-ray examination showed that the sella was enlarged and flattened near its entrance, thus making for an extra sella tumor. There were absent the fatty dystrophies, the stupor, polyuria, glycosuria and the mental symptoms together with the homonymous hemianopsia which one sees in tumors of the sella. The patient was operated on, but died soon after.

The ninth observation was of a tumor of the sella with acromegaly associated with ankylosis of the spinal column. That this patient had developed a marked acromegaly was deduced from the history, from a measurement of the hands and face and from the characteristic appearance of these and of the feet. That a tumor of the hypophysis was present was suspected from the alterations in the sella and the clinoid processes and from a complete absence of disturbance of vision, which is explained by the fact that the sella was restricted above by an approximation of the clinoid processes thus allowing for an extension of the tumor downward and therefore away from the chiasm. The importance of this case lies in the fact that the acromegaly had developed many years after the onset of the arthritis deformans spinalis. The patient had root pains, lower motor neuron disturbances and reaction of degeneration in the right lower extremity and radiographic evidences of the vertebral involvement in the lumbar region with almost complete disappearance of the intervertebral disks and rarefaction of the bodies. The author after a review of the literature has failed to find a similar case. He believes that the pains in acromegaly, referred to by authors, are due in many cases to association of this disease with arthritis deformans spinalis. He also believes that the two conditions have the same etiology, namely, a dyscrasia glandulae.

The tenth case was a tumor of the clivi sella and hypophysis. The patient began to suffer from headaches, vomiting, dizziness and mild optic neuritis followed shortly afterward by focal symptoms consisting of paresis in the third, sixth and seventh cranial nerves, with slight increase in tendon reflexes. On the right side there was involvement of the third, sixth, seventh and fifth nerves, blurring of the outlines of the disks, diminution of the senses of taste and smell, cranial percussion, pain in the right occipital region and seven months later, mental dulness and somnolence. An examination of the brain at necropsy explained the presence of various symptoms. The pressure of the tumor on the right half of the pons and pedunculus explained the paralysis of the cranial nerves, including the oculomotor. Despite the intense pressure on the right half of the bulb, there were absent any symptoms of tenth, eleventh and twelfth cranial nerve disturbance. The fact that from the beginning there were focal signs pointing to the pons, would lead one to believe that the tumor began in the distal portion of the hypophysis, extending backward and upward on the clivus, and then finally invading the entire pons.

The eleventh case was a tumor of the right rolandic area. The headache was localized in the right temporoparietal region, there was dim vision, jacksonian attacks in the face and both extremities on the left side, loss of vibration sense in the left upper extremity, gradual paresis in the left face, upper and lower extremity, and pain on percussion over the right temporoparietal region. The irritative motor signs began by preference in the left upper extremity, and then became diffused through the left side. This left no doubt as to the site of the lesion. A tumor was diagnosed in the cortex cerebri in the prerolandic gyrus of the left (typographical error, should read right) side, which later extended to the postrolandic region. In this case there was absence of vomiting and dizziness and, despite the disturbance of vision, no changes in the fundi, symptoms which are characteristic of circumscribed tumors of the rolandic area. Operation confirmed the diagnosis. No necropsy examination was allowed.

The twelfth case was a tumor of the left prefrontal lobe. The patient's condition began with nocturnal headaches, vomiting, epileptiform seizures, papilledema (bilateral), increase of albumin in the spinal fluid, tachycardia, amnesia for words—this symptom being most marked for the language which he used least—a tendency to fall to the left and backward, mild and transitory disorientation, amblyopia and paresis of both sixth nerves and of the right third with exophthalmos most marked on the left side. On the left side there was anosmia, the head being turned toward the left, exaggeration of the patellar and pain on pressure of the eyeball. On the right side there was paresis of the fifth, seventh and twelfth nerves and of the extremities, hyporeflexia of the cornea, exaggerated tendo-achillis reflex and diminution of vibration sense in the toes, painful percussion of the head, diminution of vision and adiadokocinesis. It was easy to exclude a tumor in the rolandic area because of the absence of jacksonian seizures and dissociated paralyses; of the occipital lobe, because of the absence of hemianopsia and the presence of the amnesia, pain on percussion in the frontal region and the cerebellar gait. One might suspect the cerebellum because the cerebellar gait was present early in the disease, but the other symptoms excluded this possibility. By exclusion, the site of the tumor was localized in either the temporal or frontal lobe of the left side. There is always great difficulty in making a differential diagnosis between tumors in these positions. All the symptoms which this patient presented might accompany a left temporal tumor, if one admits that it began on the surfaces of the anterior portion of the temporal lobe. Against this possibility would be the diminution of vibratory sensibility and the rotation of the head toward the left. These symptoms, however, appeared late, and for a long time the exact localization of the tumor was not possible. The fact that pain on percussion was most marked over the frontal bone of the left side was a great aid. Against this symptom the absence of motor aphasia was of little value because many tumors of the left prefrontal lobe have been reported, even those which involve the third frontal and the insula, run through their course without this symptom. The absence of this sign, however, made for the localization of the tumor in the medial rather than in the lateral portion of the convexity of the lobe in question, which is an important point for the surgeon. To him it is not a matter of indifference as to whether the medial, orbital or convex portion of the frontal lobes are involved or whether, if the latter, the superior and medial frontal gyri are affected rather than the inferior. Duret has attempted to discriminate conditions in these localities, but the author is not prepared to accept them without reserve.

This case was also important because it illustrated the fact that psychic disturbances are not always present to a marked degree in frontal lobe tumors. The patient never suffered from the "Witzelsucht" of the German authors. Müller recently desired to have this symptom taken from the category of signs of frontal lobe tumor, and Stern has asserted that the presence of this condition depends on differences both local and individual and that the condition found is not always a euphoria. The psychic complex depends on the personality of the individual and, in the average case, consists merely of mental dulling and apathy. This is illustrated in the author's case, the patient being acutely interested in his condition to the last. However, the author agrees with Stern that usually in tumors of the frontal lobe the mental symptoms occur rather early and consist of loss of attention, apathy, memory disturbances and finally, when the tumor is exerting great pressure, mental dulling. The tumor discussed by the author was extramedullary. This type of tumor is more usually accompanied by mild mental disturbances, whereas, in subcortical tumors, the mental disturbances are profound and early. Stern recalls the fact that cerebellar tumors, which are in relation to the cerebrum as extramedullary, produce but rarely any marked mental disturbances excepting the terminal mental dulling. Many authors agree to this point, namely, that subcortical frontal tumors produce early and pronounced psychic disturbances, whereas, cortical tumors are not so apt to do so. Raymond thinks that this is due to the fact that the commissural and tangential fibers of association are destroyed. The paresis of the face and extremity of the right side, in the author's patient, is explained on the theory that a mild compression of the rolandic motor area subcortically had taken place. This also explains the absence of the irritative jacksonian seizures. The fact that the right patellar reflex was diminished was due probably to an irritation of the cerebellum on the opposite side by compression, which would also explain the mild hypotonia and the transitory adiadokocinesis. There were diminution of vibratory sense and paresthesia in the right extremities for the same reason, namely, compression of the postcentral gyrus and the lower parietal lobe. Interesting, too, in the author's case was the tendency to fall to the left and backward and the ataxic cerebellar gait which has been referred to, particularly by Bruns, as occurring in frontal and temporal lobe tumors. Bruns believes that the center of the musculature of the trunk is to be found in the prefrontal lobe which controls walking in erect posture. This center controls the automatic cerebellar centers for the maintenance of equilibrium. The author agrees with this conception, although he feels that it is not always due to pressure or injury of the frontocerebellar pathways, which have indirect connection with the central nuclei of the vermis of the opposite side. In his case, the tumor being on the left side, the right cerebellar hemisphere should have been the one involved. To explain the ataxia and the tendency to fall toward the left, one must believe that the left cerebellar hemisphere was affected indirectly by compression. The author refers to the symptom of rotation of the eyes and head to the right in his patient, and recalls that at necropsy it was found that the left second frontal convolution was found completely destroyed. He recalls that Bruns has found a center in this convolution in monkeys, which governed the movements of the head and eyes toward the opposite side. It would appear, therefore, that the foot of the left second frontal convolution governs the oculo-cephalo-gyric movements of the opposite side.

In tumors originating in the basal surface of the prefrontal lobe, the ocular muscles may become paralyzed and the first branch of the trigeminus

may become the site of pain caused by compression on the cavernous sinus where the oculomotor and ophthalmic branch are in close proximity. This would explain why his patient was unable to move the eyeballs in any direction except downward. The compression of the sinuses mentioned also explains the exophthalmos and the tenderness of the eyeballs. The author discusses what he calls *amnesia verborum* and explains that his patient tended to express himself in only one of the four languages which he spoke, namely, his native language, French. At necropsy it was found that the entire area of Broca was pressed on but not destroyed, and while the transitory dysarthria is well explained by pressure on the *pars opercularis* of the third frontal and of the foot of the precentral gyrus and possibly even of the left lenticular, nevertheless, so far as the *amnesia verborum* is concerned, we are obliged to say that this was due to distant compression on the temporal lobe, destruction of which the author believes is the cause of sensory aphasia. One is led to suppose that the faculty of recalling words heard cannot possibly be localized in any special zone as many believe, but is spread through the entire frontal and temporal lobes. The recalling of a word is a very delicate constituent of the comprehension of language. Therefore, even a mild disturbance will accentuate the loss of this function. The author has frequently observed in abscesses of the left temporal lobe, that the *amnesia verborum* is an early sign and that the sensory aphasic signs soon disappear after the abscess is evacuated, but the recalling of words is the last symptom to return, being difficult of reacquisition because it is a high, associative function. The same difficulty, namely, the difficulty of recalling, is seen in the senile psychoses and in *neurasthenia cerebri*. The author ends his article by referring to the great aid which the roentgen ray gave in this last case. The plates revealed a white area corresponding to the frontal lobe on the left side, probably due to a disappearance of the internal table of the skull.

American neurologists who do not know Mingazzini are referred to the many works on neuro-anatomy and pathology which this sterling worker has written.

OSNATO, New York.

POISONING BY HYDROCYANIC ACID GAS, WITH SPECIAL REFERENCE TO ITS EFFECTS ON THE BRAIN. SAMUEL W. LAMBERT, Neurol. Bull. 11:93 (March) 1919.

The author gives a detailed description of a case of hydrocyanic acid gas poisoning, which, in view of its rarity, the meagerness of reports on the subject, and more particularly the excellent gross and microscopic pathologic studies of the brain lesions and the numerous illustrative plates, is well worth reviewing somewhat at length.

An Italian disinfecter was found unconscious after having lain for twenty minutes in a room filled with hydrocyanic acid gas. The immediate symptoms noticed were: stertorous breathing, flushed skin, engorged veins of red color, contracted pupils, nonreacting to light; abdominal muscles firmly contracted, extremities rigid, temperature, 99; pulse, 120; respiration, 36. A few hours later the pulse came down to 70, the pupils alternately dilated and contracted, he perspired freely, there were involuntary defecation and micturition and frequent muscular twitchings. The urine showed a slight trace of albumin. Blood count revealed: 6,200,000 red blood cells, 15,100 white blood cells with 90 per cent. polymorphonuclears and hemoglobin 90 per cent.

During the following days the pulse remained slow most of the time, res-

piration in the twenties, temperature varied from 99 to 104, tonic convulsive seizures set in, a doubtful Babinski reflex appeared, sweating continued, there was difficulty in swallowing, the body was rigid; then the knee jerks disappeared, as did the Babinski reflex, tonoclonic convulsions appeared several times a day. After a few days the patient came out of his stupor, recognized people and carried out some commands, at which time a fine tremor and ataxia of his arms were discovered. He could see and hear, but there seemed to be an aphasia. Three days antemortem he grew more stupid, and died sixteen days after being poisoned. At first the spinal fluid showed 207 cells, of which 45 per cent. were polymorphonuclears and 55 per cent. lymphocytes; subsequently there were only 120 cells with 60 per cent. polymorphonuclears and 40 per cent. lymphocytes.

General necropsy revealed acute parenchymatous nephritis. The brain (reported on by Dr. F. Tilney): The meninges were adherent along the edges of the superior longitudinal sinus. On macrotonic section the entire white substance was much softer than usual; numerous small hemorrhagic areas were seen in the frontal and occipital lobes, the white substance of the centrum ovale and cerebellum. Microscopically, the cortical layers were found normal in architecture. The large pyramidal cells showed cloudy swelling. In the prefrontal area there was a decided gliosis. The Betz cells showed irregular outline, cloudy swelling and eccentric nuclei. The cerebellum had the most pronounced changes in the lateral lobes and vermis. The Purkinje cells disappeared almost entirely; there was marked atrophy and irregularity of the molecular and granular layers. There were numerous small hemorrhagic areas; many rod-shaped cells of unusual character were scattered profusely, seemingly independent of the vessels. There was no neuronophagia in the cerebellum as there was in the precentral area. The most marked changes were in the Purkinje cells.

An analysis of the symptoms shows the initial coma to have been due to the temporary paralytic effect of the toxin on the motor cell. The hypertonic condition is explained by the "irritative poisoning of the cerebellum with a continuance of the original disturbance of the frontal lobes," the contracted pupils by cortical irritation, the ataxia by the cerebellar lesion, the tonic convulsions by direct toxic effect on the motor cells of the cortical area, the aphasia by the degenerative condition of the motor cells in the speech area.

Aside from the fact that the case is very unusual, it is extremely interesting to the neurologist because of the complete study of the brain lesions. Most cases are either immediately fatal, allowing little time for brain changes to occur, or else recover without leaving much damage behind. Previous clinical reports of cases which survived several days seem to have given fairly analogous pictures and no doubt had similar pathologic brain lesions. It is interesting to note that the radical, cyanogen, besides exerting specific toxic effect on the hemase of the blood, thus not allowing the freeing of the oxygen from the hemoglobin, also seems to have a special toxic effect on the Purkinje cells of the cerebellum.

The microphotographs illustrate exceedingly well the hemorrhagic changes in the various parts of the brain cortex, cerebellum and centrum ovale, and a few normal sections from the temporal cortex serve to bring out the toxic changes in the large cells of the affected parts of the brain.

WECHSLER, New York.

Society Transactions.

CINCINNATI NEUROLOGICAL SOCIETY

Regular Meeting, Sept. 25, 1919

FRANK W. LANGDON, M.D., *President*

A SPECIMEN OF BRAIN TUMOR. Presented by DR. CHARLES E. KIELY.

The patient, a boy, aged 19, was admitted to the Cincinnati General Hospital, Aug. 4, 1919. The chief complaint was convulsions, accompanied by screaming, which had first been observed in July of the same year. He also complained of diplopia. Examination showed normal fundi, slight ptosis, paresis of the superior rectus on the right, sluggishness of the right pupil to light and very slight weakness of the right peri-oral muscles. On looking to the left there was nystagmus with the fine component to the left. The result of vestibular tests was negative except for hypersensitiveness. The spinal fluid was under increased pressure, but laboratory reports were negative. The first diagnosis was disseminated sclerosis with hysteria, as many of the seizures were obviously functional and amounted merely to episodes of screaming.

In the next three weeks the patient became deaf, his whole right side weak and examination of the optic disks showed papilledema. Within another week he was lethargic and the papilledema had reached a point where the ophthalmologist advised decompression for the conservation of vision. The patient at no time had headache or vomited. Bradycardia appeared only a few hours before the operation. A sub-occipital decompression was made by Dr. John A. Caldwell. An opening in the skull about one inch wide and extending an inch and a half to either side of the median line was made. On opening the dura the cerebellum herniated into the opening and no pulsation could be detected so that the object of the decompression was not attained. The patient died twenty-four hours later and an unskillful necropsy was performed, the brain being much lacerated and distorted. There was no neoplasm in the right cerebellopontine angle, but on account of the condition of the tissues, its point of origin could not be determined. A tag of the tumor was adherent to the right occipital lobe of the cerebrum. Paraffin sections showed it to be an endothelioma.

OPERATION FOR INJURY OF THE ULNAR NERVE. Discussed by DR. JOHN A. CALDWELL.

He presented two patients who had been operated on for injury of the ulnar nerve. Both were battle casualties. The first patient had been wounded in the Argonne in October, 1918, receiving a gunshot wound of the arm on the inner side just above the elbow. He had had no primary treatment other than débridement. Dr. Caldwell had first seen him in February, 1919, at which time the patient had paralysis and atrophy corresponding to an ulnar lesion. Deep sensation was retained and there was tingling on percussion of the nerve trunk, but cutaneous sensation was gone.

Operation in March by Dr. Dean Lewis revealed the ulnar nerve embedded in scar tissue. It was freed and surrounded by a layer of fat. At the present time there is considerable improvement, but there is still marked atrophy of the muscles of the hand supplied by the ulnar nerve. Until the past few weeks he has had daily massage and electrotherapy.

The second patient received a wound from a high explosive, Nov. 11, 1918, on the inner side of the arm above the elbow, completely severing the ulnar nerve. It was sutured the following day. When the patient was seen by Dr. Caldwell in August, 1919, there was evidence of complete division of the ulnar nerve. An incision extending 4 inches above and below the elbow was made over the nerve, and it was freed from surrounding scar tissue. Longitudinal incisions in the nerve disclosed that practically the entire cross section was infiltrated with scar tissue and consequently no conduction could be expected. To remove all of the infiltrated nerve required the sacrifice of 4 inches. The proximal segment of the cut nerve was loosened from its normal bed for about one third the distance up the arm and was passed through the deep fascia to a position just lateral to the median nerve. The distal segment was freed from its normal position and passed under the pronator teres, flexor carpi ulnaris and palmaris longus to a position just to the inner side of the median. With the ends of the nerve transplanted to the flexor side of the elbow, there remained a gap of but an inch and a half. This gap was closed by flexion of the arm to an acute angle, and the nerve was then sutured. The arm was held in this position for three weeks. The angle was then changed at intervals of two weeks until after six weeks all splints were removed. At present the ulnar nerve can be palpated as a tight cord on the flexor side of the elbow when the arm is two thirds extended. Of course, no return of function is seen at this time.

Attention was called to a phenomenon that has frequently been noticed in peripheral nerve injuries. In the first case, which had simply an incarcerated nerve, there was atrophy of extreme degree of the hypothenar eminence, the adductor of the thumb and the interossei; while in the second case, in which there had been complete interruption, there was some atrophy, but it was not nearly so marked.

Book Reviews

DIE NERVOSITÄT ALS PROBLEM DES MODERNEN MENSCHEN:

Ein Beitrag zur Psychologischen Weltbetrachtung. HERBERT OCZERET, Med. Prakt. (Zürich), Zurich: 1918. Druck und Verlag: Art. Institut Orell Füssli. Pp. 95. Price, 3.80 francs.

Apparently this interesting little monograph is meant more for the laity than for the medical profession. It is in part an essay, an oration, a harangue and a disquisition. Some paragraphs are suggestive of the slang words "barnstormer" and "spellbinder." But the author appeals to one as the free-lance always does. He praises Freud, with reservations; commends Adler, but stabs him in vulnerable places; and lauds Jung, but criticizes some of his dicta.

The first part is largely a bitter impeachment of the general medical profession for its ignorance of, and indifference to, the nature of "nervousness," which the author treats as more or less of an entity.

Chapter two contains an intelligent discussion of the views of Freud, Adler and Jung. Without complete acceptance of Freud's views, the author gives him great credit for formulating a really mental treatment for mental disorders and carrying us beyond the stage of virtual electrodes and asafetida. He also commends Freud's moral courage in "rubbing under our noses" one of the weightiest social problems of the times, but intimates that an intemperate enthusiast was required for such an undertaking.

Having asserted that "nervousness" has enormously increased since 1750, the author refers it to changes in the mental attitude and emotional life of the common people, and especially to the development of self consciousness, individuality and freedom of personal feeling and action; also to the concomitant freer, more complicated and more emotional relations between men and women.

Some space is devoted to an estimate of the social value of the neurotic. A neurosis is born of nonvictorious conflict, but does not necessarily imply low value of the person (*Minderwertigkeit*). It is due to (1) complexity of problem, (2) complexity of the modern psyche or (3) ignorance of the individual ego and his lack of appreciation of necessary living conditions. On these assumptions, the author thinks that many a neurotic, like any delicately constructed and finely adjusted machine, may be one of the most valuable productive items in our modern cosmos—if properly understood and handled. He gives Bismarck as an example, a known neurotic, but a most effective and valuable citizen. However, he does not show that Bismarck was understood and wisely handled, but that the man himself had the intellect and stamina to manage himself.

Nearly half of the ninety pages of text are on the rearing of children; one case is related in detail. As a plea for closer attention and better understanding, the matter is good but there is little in the way of practical instruction.

The problem of the modern woman is given twenty-six pages, apparently because "woman as such is a nascent or rising (*emporsteigend*) class." The problem of the modern man is accorded but fourteen pages. Is it because

his song is now *diminuendo*? In the section on woman the author gives advice to both husband and wife in the form of a long letter to each containing unexceptionable, fatherly advice. The ideas are good and well expressed, the advice is sound and the decisions rational. But will either man or woman abide by them?

DIE NERVOSITÄT BEI DEN JUDEN: Ein Beitrag zur Rassenpsychiatrie für Aerzte und gebildete Laien. DR. MED. RAFAEL BECKER, Assistenzarzt der Kantonalen Heil-und-Pflege-Anstalt in Königsfelden bei Brugg, Zürich, 1919. Druck und Verlag: Art. Institut Orell Füssli. Pp. 31. Price, 2 francs.

That functional nervous disorders and mental disorders are more frequent in Jews is conceded, this being supported by the best authorities as well as by the author's personal experience. But that one may conclude therefrom that the Jews have a predisposition to such disorders, that is, "have something in them which allows them to become nervously or mentally sick more frequently than others," is warmly contested by the author.

There is presented a rather detailed consideration of the incidence of the common forms of mental disease and disorder, including epilepsy and a summary of various views on the subject, but the principal thesis of this essay is that the nervousness of Jews is due to no degeneracy but to former and present social conditions. It is a bit startling to read that "in these modern times the Jews suffer much greater torments than their forbears in the middle ages and earlier times, the times of torturing, the rack and *autos da fé*," but the author's argument on this is interesting. He says that in those days the Jew had his faith and was a joyful, even exalted martyr, whereas now he has the feeling of one who is looked down on, with no spiritual recompense.

Evidently the essay was written for Jewish readers, but it is interesting, well written and not unfair.

WAR NEUROSES AND SHELL SHOCK. FREDERICK W. MOTT, M.D., LL.D., F.R.S., F.R.C.P., Brevet Lieut.-Col., R.A.M.C. (T.), Senior Neurologist to the Maudsley Neurological Clearing Hospital; Consulting Physician, Charing Cross Hospital; Director of the Pathological Laboratory of the L. C. C. Asylums; Corresponding Member of the Neurological and Psychiatric Societies of Paris. With Preface by the Rt. Hon. Christopher Addison, M.P., Minister of Reconstruction. Pp. 348. London, Henry Frowde, Oxford University Press, Hodder and Stoughton, Warwick Square, E. C., 1919.

So far as we know, this is quite the most extensive work on war neuroses that has appeared. Apparently it aims to cover the subject in a complete and systematic way and does treat of nearly all the multifarious phases of war neuroses more fully than has yet been done in a single book. The author is an able neurologist quite exceptionally trained in the physiology and organic pathology of the nervous system and peculiarly skilled in its microscopic examination, but at the opening of the war he was obviously unprepared by his previous experience for effective and fruitful study of the psychoneuroses. Indeed, he frankly avows his earlier (quite gross) errors in the explanation of shell shock, but he still retains in this book a great deal of his earlier organic interpretation. He also introduces a considerable disquisition on surgical shock and shock in general, all of which has little to do with war neuroses, but it is very interesting and informing. It seems a bit of a pity

that this excellent monograph should not be brought really up to date by eliminating most of the author's earlier speculation produced when he was earnestly groping for the light—which he subsequently found. But elsewhere, also, the volume shows evidence of being in considerable part built up by assembling a lot of previous disjointed papers and notes.

The book begins with a classification of the effects of high explosives (*sic*) on the central nervous system: (1) the immediately fatal cases; (2) cases of wounds and injuries not fatal, and (3) affections of the central nervous system without visible external injury.

Although the author recognizes that "the psychogenic factor is by far the most frequent and important" one in the genesis of psychoneuroses and although the term "shell shock" has been officially banished from the British army, he still preserves the term, with his own personal definition. We think this unfortunate, as general use of the term has already done much harm in and out of the army. Further, the best of men disagree as to what shell shock is or ought or ought not to be. Certainly there is no general agreement with Colonel Mott when he says:

"Shell shock" is a useful term if it is limited to cases where there is definite evidence of a shell or bomb bursting near enough to knock the man down, or blow him up in the air and cause a temporary loss of consciousness."

We also doubt that all competent observers will agree to "the fact that a man who has suffered from true shell shock is not fit to return to general service for six months at least."

The book is based on an enormous personal experience and probably nowhere can be found a more complete array of cases of all kinds or a more reliable report on the microscopic changes in the nervous system to be found after severe concussion or physical commotion.

Sixteen pages are devoted to dreams and many interesting and instructive cases are cited. While the ideas of Freud and others are considered, the author seems to be scarcely at home in this field and takes the sayings of literary philosophers such as Lucretius and Shakespeare as seriously as the work of psychologists. To be sure, the exact value of the work of the latter investigators has not been determined.

Following the section on dreams is a chapter of seventy pages on war psychoneuroses, although these disorders receive more or less attention in other parts of the book. Few neurologists will seriously dissent from the opinion that "the psychopathology of war consists fundamentally in the exaggeration and preservation of instinctive defense reactions incidental to normal physiological conditions, viz., protective pain, fatigue and emotion." The various functional motor disorders are considered in some detail and under the head, "Examination of the Patient," are catalogued all the principal findings: motor, sensory, special sense, cardiovascular, gastro-intestinal, etc. The part on differential diagnosis is weak and the better portions are taken bodily from the French. On page 139 it is stated that "hysterical hemiplegia with contracture or hysterical hemicontracture develops all at once." Any one familiar with hysteria knows that this may sometimes be true but frequently is not.

The author's attitude regarding Babinski's ideas on physiopathic or reflex disorders is not exactly clear, but he seems to give them a somewhat half-hearted endorsement.

There are a couple of pages on "The Mental Conflict in Relation to War Psychoneuroses." Many readers will consider them quite inadequate. Per-

haps they were written carelessly, for they seem to say that such conflicts are limited to officers, which is, of course, perfectly absurd.

Thirty pages are devoted to gas poisoning, principally by carbon monoxid. A large part of this matter is old, but it is exceptionally good and of value in civil as well as in military conditions. Other interesting and instructive things in the book are a consideration of windage and its effects, voltaic vertigo, shock in relation to memory (including musical memory) and hysterical speech defects.

The section on treatment (30 pages) is far from complete but well balanced and reasonable. It will be of great assistance to the general practitioner and contains hints of value to the expert. The consideration of war psychoses is very sketchy but may suffice for those not especially interested in the subject.

The bibliography is decidedly inadequate, the index excellent; the illustrations (eighty-one in number, of which three are in colors) are very good indeed, and printing and binding are entirely satisfactory.

It is a good book though it has some pretty obvious defects. It is quite the best that the general practitioner can get and one that no general neurologist or psychiatrist can quite afford to be without.

PSYCHOSES OF THE WAR, INCLUDING NEURASTHENIA AND SHELL SHOCK. H. C. MARR, M.D., Lieut.-Col., R. A. M. C. (Temp.); Fellow of the Royal Faculty of Physicians and Surgeons, Glasgow; Neurological Consultant to the Scottish Command; H. M. Commissioner of Control for Scotland; Formerly Specialist in Nervous Diseases to the Troops in Malta (1915-1916); Physician Superintendent of Glasgow District Mental Hospital, Woodlee, Lenzie; Referee, Under the Workmen's Compensation Act for Dumbartonshire; and Mackintosh Lecturer in Psychological Medicine, St. Mungo's College, Glasgow, etc. Pp. 328. London, Henry Frowde, Oxford University Press; Hodder and Stoughton, Warwick Square, E. C., 1919.

In reading this work for review a sympathetic attitude becomes more and more difficult because, as one proceeds, evidence accumulates of a fine opportunity lost and of much valuable clinical material gone to waste. Having made this somewhat caustic declaration, the reviewer feels obligated at least to indicate his justification.

Discontent begins with the Introduction, which is a loosely written mixture of anatomy, physiology, pathology and clinical psychiatry, in which the known is not well discriminated from the hypothetical and relative values are not in accord with modern medicine. Under "Means of Observation of Mental Phenomena," the author stresses almost exclusively facial expression, a sort of sublimated physiognomy, properly and decently buried years ago.

In Chapter 2 the author indicates that shell shock and neurasthenia are the same thing, a subhead being "Neurasthenia (Shell Shock)," and states that "hysteria is in reality a symptom complex of neurasthenia." Having agreed with Charcot that hysteria is a mental condition, in the next sentence it is "a functional affection of the cortical, bulbo-spinal and sympathetic neurones." Nowhere does one find a definition of neurasthenia, but the author's division of it into (1) simple; (2) hysterical, and (3) organic is sufficiently inclusive. Under "Neurasthenia of Toxic Origin" he alludes to a frequent expression of soldiers exposed to shell explosion in a confined place and draws certain conclusions therefrom. The soldiers say the explosion has "taken the

guts out of them" and therefore Dr. Marr concludes that "these results can only be ascribed to the effect of concussion on the abdominal sympathetic nervous system." An illustration on page 52 bears the legend, "Facial Expression of Fear in Hysterical Depression (Toxic Neurasthenia)," and on page 69 we read: "*Observation.—Hysterical neurasthenia from bacillary infection; mental confusion and emotionalism. Exciting cause; shell explosion. Recovered after use of autogenous vaccines.*" Did the shell explosion cause the bacillary infection? The account of the case does not answer the question. Nor is it made clear whether the vaccines or the sinusoidal baths (also used) cured the patient or whether he wearied of both and just naturally got well.

Observation 6, in the section on malingering, is labeled, "Simulation of deaf-mutism in neurasthenia," and the text says the patient "really suffered from hysterical deaf-mutism." Does this indicate loose classification or careless writing or slovenly thinking?

Again, one is struck by the satisfied conclusiveness with which the author introduces a diagram of brain centers and association tracts to make clear the phenomena of sleep and dreams. But it must be conceded that to diagram is much easier than lucidly to explain.

Ordinary phobias are classed as "insane obsessions." Janet's psychasthenia is made to carry some long since discarded ideas of the older psychiatry as well as forced to include "mental rumination" in the category of "forced spasmodic movements." On pages 132 and 133 patients who present obvious constitutional inferiority, not to say high grade imbecility, are cited as psychasthenics.

The book abounds with interesting illustrative cases, but essential details are frequently lacking and the reader must make his own interpretations; those of the author seldom are very helpful.

In approaching the subject of treatment of the psychopathies such expressions as "undermined the nervous system and the inherent constitution of the neurones," and "all external influences which irritate the wounded mind," would lead one to expect some glittering generalities; and they are there. But the author is sufficiently explicit in recommending Easton's syrup, the effluve from a Wimshurst machine, and sinusoidal baths—especially the last. To be sure he is not clear as to whether the quite remarkable effects of the baths are due to suggestion or because they "act directly on the circulatory system by causing a relaxation of high arterial tension." Nor does he even hint how relaxation of high arterial tension really acts on the circulatory system nor how, this action having been attained, the psychopathy is affected by it.

One hundred and three pages are devoted to mental deficiency, toxic psychoses and organic psychoses, though of course no particular relation of these affections to the war is shown, except some statistical statements as to their relative frequency. In these pages the reviewer has been able to find nothing new of value but quite a bit that generally has been discarded. Here, too, is found considerable looseness of classification and vagueness of outline, not to mention some careless use of the English language which contributes its mote to the prevalent lack of clarity.

The book closes with thirty-four pages of an elaborate scheme of "mental case-taking." The scheme contains some excellent and practical ideas and should be consulted by any one formulating a plan of record for an institution or for some special purpose.

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